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MALIGNANT TUMORS OF THE MIDDLE EAR AND THE MASTOID PROCESS

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Any malignant neoplastic process involving the middle ear and the mastoid process has generally been considered almost a pathologic curiosity. Moreover, the condition has been almost universally supposed to respond unfavorably to therapeutic measures. Actually, it occurs less rarely than the medical literature would lead one to believe, and if it is recognized reasonably early and is dealt with adequately, the prognosis is as satisfactory as that generally offered by cancers of the nasal accessory sinuses.

LITERATURE

The literature on malignant tumors of the middle ear and the mastoid process is not extensive. The largest series of cases reported (13) is that of Thorell,[1] presented with a consideration of the treatment of this condition at Radiumhemmet. Outstanding articles on this subject in the American literature have been written by Furstenberg,[2] Schall[3] and Spencer.[4] In addition, reports of cases have been presented by Robinson,[5] Bowman,[6] Smith,[7] Newhart,[8] Stokes,[9] Fraser[10] and others.

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2. Furstenberg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **33**:677-689 (Sept.) 1924.

3. Schall, L. A.: Neoplasms Involving the Middle Ear, Arch. Otolaryng. **22**: 548-553 (Nov.) 1935.

4. Spencer, F. R.: Malignant Disease of the Ear, Arch. Otolaryng. **28**:916-940 (Dec.) 1938.

5. Robinson, G. A.: Malignant Tumors of the Ear, Laryngoscope **41**:467-473 (July) 1931.

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(Footnotes continued on next page)

INCIDENCE

During the last twenty years (Jan. 1, 1922 to Dec. 31, 1941) there were seen at the Mayo Clinic 48 patients who had cancer involving the middle ear and the mastoid process. More than 500 additional patients observed during this period had cancer involving only the pinna or the external auditory canal. The incidence of malignant tumor of the middle ear and the mastoid process among registrants in the clinic during the last six years has been approximately 0.003 per cent.

This study is based on a group of 38 cases in which treatment was carried out in the clinic; excluded were 10 additional cases in which

Fig. 1.—(*a*) Recurring basal cell and squamous cell epithelioma, grade 1, measuring 3.5 by 8 cm., in a man aged 54 years. The lower portion of the lesion was ulcerated deeply, involved the auricular cartilage and clinically appeared to be invading the mastoid process. Removal was effected with electrocoagulation and interstitial application of radium. It was necessary to sacrifice the lobe of the ear and a large portion of the concha. Neither the middle ear nor the mastoid process was involved. The case was not included in the series on which this report is based. (*b*) Result six and a half months after treatment. At the time of writing the patient has been well more than three years.

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7. Smith, C. H.: Primary Carcinoma of Middle Ear and Mastoid: Endaural Approach and Mastoid, Laryngoscope **50**:547-554 (June) 1940.

8. Newhart, H.: Primary Carcinoma of the Middle-Ear: Report of a Case, Laryngoscope **27**:543-555 (July) 1917.

9. Stokes, H. B.: Primary Malignant Tumors of Temporal Bone, Arch. Otolaryng. **32**:1023-1030 (Dec.) 1940.

10. Fraser, J. S.: Malignant Disease of the External Acoustic Meatus and Middle Ear, Proc. Roy. Soc. Med. (Sect. Otol.) **23**:71-77 (June) 1930.

the process of disease was so extensive that therapeutic measures would obviously have been of little avail. Excluded also was 1 case in which the clinical and operative findings, together with the subsequent course, failed to substantiate a histologic diagnosis of epithelioma made on tissue removed from the external auditory canal and from the mastoid antrum at the time of operation. The patients were divided equally as to sex, there being 19 males and 19 females. The ages of the males ranged from 27 to 71 years and those of the females from 3 to 80 years. Nineteen patients (50 per cent) were between 50 and 70 years of age; 30 patients (79 per cent) were between 40 and 70 years. Five patients were less than 40 years old, and 3 patients were 70 years or older. The left ear was involved in 22 instances; the right, in 16 (fig. 1 *a* and *b*).

SITE OF ORIGIN

The middle ear and the mastoid process may be involved by a malignant neoplasm arising primarily in one or the other of these situations, or they may be invaded secondarily by a neoplasm that has originated in the pinna or in the structures adjacent to it. For convenience we shall designate growths of the former origin as intrinsic growths and, because it frequently is impossible to determine whether the pathologic process has developed in the middle ear or in the deeper portion of the external auditory canal, growths springing from the latter region are included in this category. Tumors having their origin in the pinna, in the skin of the surrounding region, in the parotid gland, in the nasopharynx or in other structures outside the ear are referred to as extrinsic. In our experience the intrinsic growths outnumber the extrinsic, and this predominance appears to hold generally in the cases of this type reported by other observers. Of the 38 neoplasms in this series 25 were considered intrinsic; the remaining 13 were extrinsic.

SYMPTOMATOLOGY

The clinical course of cancer of the middle ear and the mastoid process varies greatly, and the symptomatology is accordingly not the same in all cases. Among factors influencing the course and thereby the symptoms complained of are the site of origin of the neoplasm, the activity and the extent of the growth, the presence of inflammation, trauma, the treatment and the age and the general condition of the patient. In the presence of inactive cancer about the pinna or the canal that has not become infected or has not been subjected to trauma, symptoms frequently are mild, and the lesion may be present for years without causing the patient sufficient trouble to bring him to a physician. On the other hand, a highly malignant neoplasm, either intrinsic or extrinsic, is likely to produce severe symptoms promptly, especially if subjected to trauma or to incomplete treatment. Hence, previous to examination, symptoms may have been present for only a few weeks or

months, or they may have been noted for some years. In general, symptoms had been present for a much shorter period in the group of cases of intrinsic cancer than in the group of cases of extrinsic cancer. As might be expected, some of the inactive extrinsic growths produced the mildest symptoms and were of the longest duration.

Chronic otorrhea, often of many years' duration, may precede the development of a malignant neoplasm in the middle ear. This discharge frequently renders it difficult, if not impossible, to determine even the approximate date of the onset of the symptoms caused by the cancer. In a few of the cases of intrinsic cancer, polyps had been removed from the external auditory canal on repeated occasions during the course of several years preceding the examination at the clinic, but in most instances microscopic studies of the excised tissue had not been carried out and the approximate time of inception of the malignant process could not be determined. In only 4 of the 25 instances of intrinsic malignant neoplasm did it appear likely that the cancer had been present for as long as five years. One patient, a woman aged 47 years, had had a chronic discharge from the ear since the age of 10. Eight years prior to her coming to the clinic a polyp had been removed from the external auditory canal, a diagnosis of "tumor" had been made after microscopic examination and radium had been applied. Tissue removed at the clinic proved microscopically to be squamous cell epithelioma, grade 4. It is impossible to conceive of such a highly malignant neoplasm having been present for more than a few months. In 11 of the 13 cases of extrinsic growth, carcinoma had apparently been present for more than five years, and in 7 of these, for ten years or more. The longest period of duration was seventeen years; the patient was a man 50 years of age who had adenocarcinoma of the mixed tumor type originating in the parotid gland and only in recent years involving the mastoid. All of the remaining extrinsic neoplasms of long duration were diagnosed as adenocarcinoma or basal cell epithelioma.

The symptoms most commonly complained of by the patients are pain, discharge from the ear, ulceration in or about the ear and decreased hearing on the affected side. Bleeding from the ear, vertigo, facial paralysis and the presence of a tumor are encountered less frequently. Tenderness in the region of the mastoid process, tinnitus, ankylosis of the mandible and difficulty in swallowing and speaking due to paralysis of the soft palate, pharynx and tongue may be causes of complaint. The complaint most commonly made by patients in our series was of pain in the ear. A few patients, however, experienced no pain whatever. In a number of instances, pain was present also in the mastoid, temporal, occipital and frontal regions, or it was referred entirely to these parts. It was usually of a steady aching character but occasionally became lancinating and in some cases was more of a sense of pressure or throbbing than intense distress. At times the pain was severe, and at intervals

of a few weeks acute pain occurred, lasting for periods of a week or ten days. A few patients noted that their pain was constant but was worse at night and was aggravated by lying down, by moving the ear and by cessation of the aural discharge. In some instances it came on at about the same time each day.

Next to pain in or about the ear, complaint was made most commonly of discharge from the ear. This was present in 22 of the 38 cases. At times it had had its inception in acute otitis media in childhood and thus had preceded other symptoms by years. Often there was nothing unusual about the character or the amount of the discharge but at times its foul odor, watery consistency and tinge of blood had been noted by the patient. A fifth of the patients had experienced spontaneous bleeding from the canal. The number of patients in whom each of the more common symptoms was encountered is shown in table 1.

Among the 13 patients who had extrinsic cancer the presence of an ulcer or a tumor in the external canal or about the pinna or in the region of the mastoid process was the most frequent cause of complaint. In a

TABLE 1.—*Symptoms Complained of Most Frequently by Thirty-Eight Patients*

Symptom	Patients
Pain	33
Discharge	22
Ulceration	15
Deafness	12
Bleeding	8
Facial paralysis	7
Vertigo	6
Mastoid tenderness	3
Tinnitus	3

number of cases radical treatment had been carried out previously, so that the typical appearance of the neoplasm had been lost and a clinical diagnosis could not be made immediately. Extensive ulceration was present in some of the cases, and in an occasional case an ulcerated lesion was fixed to the mastoid process, had perforated the pinna or was destroying a varying portion of the auricle. In 1 instance the entire pinna had been destroyed by the ulcerative process.

The duration of the aural symptoms prior to the patient's coming to the clinic varied a great deal, depending on the nature of the lesion, its activity and the site of origin. It was longest in the cases of adenocarcinoma of the mixed tumor type and especially in those in which the neoplasm arose outside the ear. It was shortest in cases of the more active squamous cell epithelioma, particularly in those of the intrinsic group. Next to the cases of adenocarcinoma in duration of symptoms were the cases of basal cell epithelioma.

ETIOLOGIC FACTORS

In a large percentage of the reported cases of malignant tumor of the middle ear and the mastoid process the cancer was preceded by

otorrhea of long duration. This is a condition which is considered generally an important etiologic factor in the development of cancer in this region, although whether it has any direct bearing in this regard is debatable. It seems logical to assume, however, that the constant aural discharge might well cause cancerous change in the tissues irritated, just as habitual moistening of the lips by the tongue had appeared at times to initiate labial epithelioma. It is possible only to theorize in this connection, for other factors must play an important role, as is evidenced by the fact that tumors of this type frequently develop entirely independently of such irritation.

In the series of 38 cases studied there had been long-continued aural discharge in only 11. It is of interest that in 6 of these 11 cases the site of origin of the malignant process was apparently in the external canal, whereas in at least 2 of the other 5 cases it originated within the tympanum. In the remaining 3 instances the growth was so extensive that the point of its inception could not be ascertained. A number of

TABLE 2.—*Clinical Findings in Thirty-Eight Cases of Cancer of the Middle Ear and the Mastoid Process*

	Cases
Involvement of external auditory canal	28
Discharge	22
Tumor in canal	15
Obvious cancer	15
Ulceration	15
Fixation of lesion over mastoid process or to canal wall	10
Facial paralysis	7
Perforation of pinna	4
Tenderness over mastoid process	3

cases of extrinsic cancer were included in this series although the presence of a chronic discharge in association with the lesion probably is of little significance. In 6 of the 11 cases of cancer developing in the presence of persistent otorrhea the neoplasm was squamous cell epithelioma; in 3, hemangioendothelioma; in 1, adenocarcinoma, and in 1, fibrosarcoma. Other conditions encountered having a possible causative bearing were ichthyosis of the canal, occurring in a single case, and chronic pruritus of the external canal, present for twenty-five years, in another. Two patients had experienced repeated attacks of erysipelas, and in one of these patients malignant change had developed subsequently in an ulcer persisting after the acute illness.

CLINICAL FINDINGS

The clinical picture presented by patients with cancer of the middle ear and the mastoid process varies greatly depending on whether the neoplasm has originated within these structures or whether it has originated externally and is invading them secondarily (table 2). Tumors developing intrinsically may reveal little evidence of their pres-

ence until well advanced, in most instances the subjective symptoms being out of all proportion to the physical findings. In an unusual case of this type, that of a woman 43 years of age, the only objective sign was tenderness in the tissues over the mastoid process, yet exploration revealed an extensive highly malignant neoplasm of this region with considerable destruction of bone diagnosed as squamous cell epithelioma. More commonly the external canal is filled with polyps, which frequently are recurring and may be fairly fibrous. A highly vascular, readily

Fig. 2.—Adenocarcinoma of the mixed tumor type, involving the ear and the mastoid process. Note the fulness in the parotid and mastoid regions. The tumor was removed through a postauricular incision; although its outer portion was encapsulated, it was infiltrating in its deeper portion; excision was supplemented with implantation of radium; there had been no recurrence eight and a half years later.

bleeding mass or a granular growth may be present in the canal or the canal may be markedly narrowed by diffuse thickening or infiltration of its walls. A pulsating profuse foul discharge which is at times watery and blood tinged may be associated with these findings (fig. 2 *a* and *b*).

In cases of extrinsic cancer with invasion of the tympanum and the mastoid process the clinical findings are likely to be pronounced unless

treatment has been carried out recently; in a high percentage of cases the nature of the condition will be obvious to a physician who has had appreciable experience with malignant neoplasms. A clinical diagnosis of epithelioma was made in a large proportion of the cases of extrinsic neoplasm in this series and subsequent microscopic studies corroborated this determination. In a few cases the neoplasm was recognizable on inspection as carcinoma of the mixed tumor type. Cancerous ulceration of varying extent was present in most of the cases of epithelioma, and in a number there was fixation of the ulcer over the mastoid process. Direct extension of the neoplasm inward along the external auditory canal was encountered usually, although at times the ulceration overlying the mastoid process was penetrating into the bony structures

Fig. 3.—(*a*) Recurring basal cell and squamous cell epithelioma, grade 1, involving the left ear and mastoid region in a woman aged 59 years; (*b*) perforation of pinna by the lesion. Treatment consisted of electrocoagulation, radical mastoidectomy and irradiation. (*c*) Result two and a half years later. The patient had temporary postoperative facial paralysis. At the time of this report she has been well three and three-fourths years.

beneath. Perforation of the pinna had occurred in a number of cases, and varying portions of the auricle had been destroyed. In 1 case there was complete loss of the pinna (fig. 3 *a*, *b* and *c*).

PREVIOUS TREATMENT

All but 2 patients in this series had received treatment for aural disease prior to coming to the clinic. This treatment had ranged from the use of ear drops and the local application of antiseptic solutions, ointments and caustics to removal of polyps, excision of the tumor, irra-

diation of the tumor and mastoidectomy. Roentgen rays had been used in 12 cases and radium in 13. Most of these patients had had extrinsic lesions. Polyps had been removed from the external auditory canal in 7 cases, but in few of these had the tissue been subjected to microscopic examination. Mastoidectomy had been performed in 6 cases, and in 3 of these the cancerous nature of the condition had been recognized. Three of the patients had undergone mastoidectomy at the clinic from two to sixteen years prior to our establishing a diagnosis of cancer. One of these patients had had two operations on the mastoid ten and sixteen years, respectively, prior to recognition of the malignant

Fig. 4.—(*a*) Right-sided facial paralysis produced by fibrosarcoma, grade 4, of the middle ear, external auditory canal and nasopharynx. The patient is shown as she appeared almost seven years after eradication of the neoplasm by means of mastoidectomy, use of radium and roentgen therapy. (*b*) Result following correction of the facial paralysis by insertion of fascia lata loops and plastic.

growth. At the time this paper is written this patient and another have been well for periods of eight and ten years, respectively, while the third has not had recurrence after two years (fig. 4 *a* and *b*).

ROENTGENOGRAPHIC STUDIES

Roentgenograms of the mastoid process should be made in all cases of chronic infection of the ear and in those in which there is a question regarding the possibility of a neoplasm in this situation. This is particularly true in cases of an intrinsic lesion since in these the roent-

genograms frequently will furnish considerable information that cannot be obtained from physical examination alone. Roentgenographic studies are also essential in cases of an extrinsic tumor that extends into the external auditory canal or is fixed to the mastoid process. Although roentgenograms at times fail to reveal early involvement of bone by a neoplasm of this group they often help in determining the extent of the process if invasion of bone is present. Since in cases of intrinsic neoplasm the process usually originates within the tympanic or the mastoid cavity roentgenographic evidence of destruction of bone would be expected in a higher percentage of cases than in the extrinsic group. As a matter of fact, the contrary was noted in our series. Among the 25 cases in which the cancerous process arose within the mastoid cavity or the middle ear or deep in the external auditory canal, the roentgenograms revealed evidence of pathologic change in the mastoid process in 21 (84 per cent). Destruction of bone was evident in 8 cases (32 per cent), cloudiness in 7 (28 per cent), sclerosis in 7 (28 per cent) and a postoperative defect in 4 (16 per cent). Among the cases in which the primary involvement was in the pinna or the tissues adjacent to it and in which the middle ear and mastoid process were invaded secondarily roentgenograms were positive in 9 (69 per cent). Destruction of bone was shown in 6 cases (46 per cent) and cloudiness in 5 (38 per cent). In 4 cases (31 per cent) some cloudiness of the mastoid cavity was present, but destruction of bone could not be discerned.

TREATMENT

In the past the treatment of malignant tumors of the middle ear and the mastoid process has been discouraging. Such tumors are encountered so infrequently that it has been difficult to develop an effective method of therapy based on accumulated experience. Treatment has consisted chiefly of surgical removal and irradiation of the region either alone or combined, and in only the exceptional case has this resulted in more than temporary palliation. Several factors have contributed to this unfavorable response to treatment. Among these are: (1) the delay in establishing a diagnosis, (2) the difficulty of access to a tumor involving the inner portion of the external auditory canal, (3) the frequent impossibility of determining clinically the extent of involvement, (4) the timidity of the surgeon with regard to carrying out radical measures and (5) the ineffectiveness of radium and roentgen rays in controlling malignant neoplasms that are invading either osseous or cartilaginous structures. These various factors were concerned in the unfavorable results observed in a number of the earlier cases in this series, and attention to them has greatly improved the outlook.

Originality is not claimed for the method of treatment that we have employed in cases of this type. The procedure has evolved pretty

largely from that used in dealing with malignant neoplasms of the nasal accessory sinuses and larynx and, like the latter, has been improved considerably since the development of electrosurgical methods. In the earlier cases treatment was almost exclusively by placing of radium packs and by direct application of radium tubes or a plaque with or without excision. This method of treatment has been supplanted by the more rational and effective procedure of exposing the tumor surgically, removing it with electrocoagulation under direct observation and supplementing this with the implantation of radium points directly into the wound and the external application of radium packs or roentgen radiation. The results obtained with this form of treatment well bespeak its effectiveness. An exceptionally lucid exposition of the problems involved and the indications for treatment in these cases was presented by Furstenberg, in 1924, in reporting a case of primary carcinoma of the middle ear and the mastoid process. We quote from his paper:

> In the department of otolaryngology of the University of Michigan the following principles are rigidly adhered to in the surgical treatment of malignancy. First, radical excision of the neoplasm and removal of all that kind of tissue where it has originated or invaded and second the employment of a technic that will leave the site of the new growth open to inspection for the remainder of the patient's life. The radical mastoid operation lends itself to these arbitrary requirements in that it permits of radical excision of the growth and leaves the middle ear and mastoid cavity accessible to future cauterization in the event of recurrence.

Surgical Procedure.—The wide removal of the accessible portion of the neoplasm by means of electrocoagulation, either the coagulating current or the cutting current or both being employed, seems definitely to be an improvement over excision. Often the tumor is fixed to the mastoid process and must be stripped directly from the bone. If there is any question regarding the possibility of the bone being invaded, the involved portion should be removed and the mastoid process opened. Perforation of the pinna may be present, in which event it is necessary to sacrifice enough of the full thickness of this structure to include a wide margin of the normal tissue. When, as is commonly true, the growth fills the external auditory canal or extends deeply along its walls, electrocoagulation is carried as far as it is possible to visualize the tumor. The mastoid process then is opened, and if neoplasm is not encountered, the procedure at once is carried through to completion as radical mastoidectomy, the posterior wall of the canal being taken down. If tumorous tissue is encountered on removal of the bony cortex, which is often the case, this tissue is destroyed with the diathermy electrode, the dissection of bone being continued as necessary to expose more of the neoplasm. In this way electrocoagulation and dissection of bone are alternated until the tumor has been completely

removed. During this process a varying portion of the dura may be exposed. In fact, a wide area of dura and a considerable part of the lateral sinus, the jugular bulb and other intracranial structures may be uncovered. At times an extension of the tumor is found directly in contact with the dura or sinus, and there may be malignant infiltration of these parts. When this is true, the possibility of effecting complete removal of the neoplasm is practically nil. Fortunately, much more often, even though the tumor is highly malignant, a thin capsule will be found separating it from the dura and, provided the remainder of the growth can be cleanly removed, the primary one can be entirely eradicated. As the bony posterior wall of the external auditory canal is removed, if this wall has not already been destroyed by the pathologic process, the portion of the tumor that could not be reached through the meatus is rendered accessible.

At times a tumor that is well encapsulated on its outer surface will be found infiltrating in its deeper portion. The contrary may also be true. Both of these contrasting conditions have been seen in cases of adenocarcinoma of the mixed tumor type in this situation. Provided the dura is intact, surprisingly little postoperative reaction usually follows the surgical procedure even though the neoplasm has been extensive and has necessitated intensive electrocoagulation. There is no doubt that in questionable cases the risk of meningitis and of other intracranial complications is increased greatly by attempts to deal with these conditions more conservatively and by failure to open the mastoid process and to expose the dura. After surgical intervention the patient as a rule experiences marked relief from pain, and if the operation has been complete, cessation of the otorrhea is phenomenal. However, if the neoplasm is infiltrating the dura, any relief from symptoms produced by removal of the bulk of the tumor is likely to be only transitory, and prompt recurrence is almost sure to follow even though the operation is supplemented with intensive irradiation of the site (fig. 5 *a, b, c* and *d*).

It has been our routine after surgical removal of a malignant neoplasm of the middle ear and mastoid process to place radium points or tubes directly in the operative cavity and to give a fairly caustic dose. If removal of the tumor appears to have been clean, points containing 1 mg. each of radium element encased in platinum usually are employed, from ten to fifteen of these being held in place by means of iodoform gauze, which keeps them well removed from the bone itself. These points remain in the wound for from twenty to thirty hours, a total dose of 300 to 400 mg. hours being administered. A few days after operation, this treatment is supplemented with radium packs, an additional dose of from 4,000 to 18,000 mg. hours being given. When the mastoid process is opened, if the tumor is found to be highly malignant and the involvement so extensive that complete surgical removal appears impracticable, no attempt at complete surgical exenteration is made.

Instead, radium points or tubes are immediately implanted directly into the growth, and the wound either is sutured loosely or a gauze pack is inserted to hold the radium in place. The latter procedure has at times been found necessary in cases also in which extensive highly vascular hemangioendothelioma is encountered on exploration of the mas-

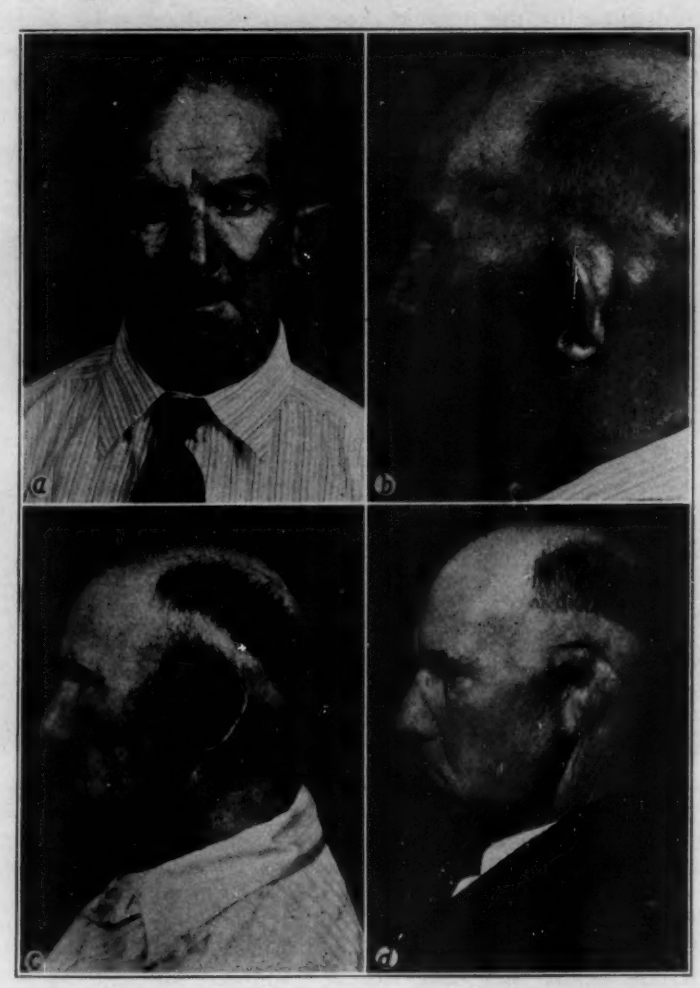

Fig. 5.—(*a*) and (*b*) Recurring basal cell and squamous cell epithelioma, grade 1, of the left ear and mastoid region. Note the neoplastic infiltration of the pinna and the protrusion of the tumor from the meatus. (*c*) Extensive defect resulting from treatment consisting of electrocoagulation, radical mastoidectomy and irradiation, required to remove the tumor. Sacrifice of the facial nerve and of the entire auricle was necessary. (*d*) Complete healing. At the time of this report the patient has been well one and a half years after treatment. No neoplasm was demonstrable in the middle ear and the mastoid process; accordingly this case is not included in the series but is presented to demonstrate the radical treatment at times required to control malignant tumors in this situation.

toid process. In some of the cases in our series the bleeding was uncontrollable and it was necessary to terminate the operation. Accordingly, insertion of radium and packing or suturing of the wound was carried out.

Considerable necrosis of bone usually results from electrocoagulation and irradiation. In the mastoid process the resultant sequestrum separates slowly, requiring a much longer period than that necessary after similar treatment inside the mouth. Whereas sequestrums about the jaws usually separate and can be lifted out readily within two months, those involving the mastoid process and the temporal bone frequently require a year or longer. Because it often is impossible to determine

Fig. 6.—(*a*) Scarring and defect present two years after removal of recurring adenocarcinoma, grade 2, of the right ear and mastoid region by means of electrocoagulation, radical mastoidectomy and irradiation. Approximately one year subsequently the patient returned without local recurrence but with involvement of three right cervical lymph nodes, for which a block dissection was done (*b*).

the extent of the necrotic process, it is inadvisable to attempt to remove sequestrums before they have loosened, for excessive trauma is likely to result from too early removal, and there is a possibility of producing a severe acute inflammatory reaction with extension of the necrosis and even meningitis. On the other hand, if removal is delayed until separation is complete, the process is found well walled off, there is no appreciable trauma and epithelization of the wound takes place promptly, even though a wide area of dura may be uncovered. Should evidence of recurrence of the neoplasm appear at any time after the operation, however, removal of the sequestrum and biopsy may become necessary even though the dead bone still is fixed firmly (fig. 6 *a* and *b*).

PROGNOSIS

In cases of cancer of the middle ear and the mastoid process the malignant process extends by direct contiguity; metastasis rarely occurs, and dissection of lymph nodes is not indicated unless these become involved. Extension into the cervical lymph nodes took place in only 2 cases in the series under consideration. In one of these cases a neoplasm diagnosed as basal and squamous cell epithelioma, grade 1, had been present for years and recurred repeatedly prior to its development in the glands. In the other case a growth diagnosed as adenocarcinoma (cylindroma type), grade 2, had been removed at the clinic on two occasions. There was no local recurrence of the malignant process at the time the patient returned with several metastatic nodes in the neck two and a half years after the last operative procedure. Block dissection was carried out.

The result likely to be obtained in any given case of cancer of the middle ear and the mastoid process is difficult to anticipate because of the great variation in the factors involved. The nature of the neoplasm, its activity and extent are extremely important, and frequently it is impossible to determine these until the mastoid process and the tympanum are explored. Many times the neoplasm will be found so extensive and highly malignant that palliation only can be expected. On the other hand, a growth that has appeared clinically to be bordering on inoperability may be found on exploration to be readily removable. This has been noted especially in cases of adenocarcinoma of the mixed tumor type. In our experience one of the most frequently encountered intrinsic growths is the hemangioendothelioma which, although it usually is of a low grade of malignancy, is so highly vascular that clean surgical removal often is impossible. In fact, in many cases electrocoagulation will not control the bleeding and coagulation cannot progress because of it. Although the results in 4 of the 7 cases of hemangioendothelioma included in our study have been satisfactory to the time of writing this report, it seems certain that a better chance of entirely clearing up the pathologic condition would have been afforded had it been possible to remove the growths by electrosurgical procedure and then to supplement this operation with irradiation of the region rather than to depend entirely on implanted radium and radium packs or roentgen therapy, as was necessary.

The combination of therapeutic measures now carried out in all cases of this type in which the extent of the tumor and the condition of the patient do not contraindicate, consisting of radical mastoidectomy, electrocoagulation and irradiation, is considered decidedly superior to the surgical excision and irradiation or irradiation alone previously employed. Good results were obtained with radium and the roentgen rays a few times, as in a case of fibrosarcoma, grade 4, of the external

canal, middle ear, mastoid antrum, nasopharynx and nasal fossa with an associated jugular foramen syndrome and unilateral facial paralysis. The patient was a woman 38 years of age. After radium packs and roentgen rays had been used, as well as radium on an applicator in the nasopharynx, the neoplasm entirely disappeared, and a plastic operation to correct the deformity incident to the facial paralysis was carried out

Fig. 7.—Patient well eight and a half years after treatment for hemangioendothelioma, grade 2, of the right middle ear and mastoid region. Residual paralysis of the right side of the face developed spontaneously.

some six years later. In general, however, palliation only is to be anticipated with this form of treatment in such cases. Even surgical removal of the growth supplemented with irradiation of the site without radical mastoidectomy does not offer the patient the chance of cure that is his with the therapy now employed. Although some of the patients in our series who had cancer of the middle ear and the mastoid

process lived for more than eight, and 1 patient for even fifteen, years after they first were treated at the clinic, they had repeated recurrences and eventually succumbed to the malignant process. The results in several such instances are not considered satisfactory, for in retrospect and in the light of experience in more recent similar cases it seems likely that had radical mastoidectomy, electrocoagulation and irradition been carried out, some of these patients would have had an excellent chance of complete cure. Moreover, the 2 patients in the series who died of meningitis and septicemia after surgical operation might well have been saved had the mastoid antrum and the tympanum been exenterated, the dura uncovered and chemotherapy employed (fig. 7).

RESULTS

A summary of the results obtained in the 38 cases in this series in which treatment was given is presented in table 3. It will be noted that 20 patients lived two years or more after treatment, 15 three years or more, 10 seven years or more, 8 eight years or more, 3 more than ten years and 1 fifteen years.

We have been unable to obtain information from 2 patients in the series since they returned home after their original treatment at the clinic. It seems likely that one of these patients, a Negro 42 years of age, may be well, as his neoplasm, squamous cell epithelioma, grade 1, appeared to have been cleanly removed. The other patient, a woman 80 years of age, had extensive squamous cell epithelioma, grade 2, and in all probability the radium packs employed produced only temporary palliation. It has been impossible to trace a third patient since her sequestrum was removed at home four months after dismissal from the clinic. In 6 cases the malignant process was extensive and palliation only was expected. All of the 6 patients succumbed to the disease within a few months to four years, but it is interesting that one of these patients was carried along for four years by means of radium packs even though at the time of mastoidectomy the squamous cell epithelioma, grade 1, which filled the mastoid cells also involved the dura. At the time this paper is written 2 patients probably have recurrences, one after eight months, the other after fourteen months. One patient was living and free from adenocarcinoma of his middle ear eight and a half years after removal of his neoplasm. Death, which occurred more than ten years after operation, probably was not due to the carcinoma. Another patient who had recurring adenocarcinoma that appeared to be bordering on inoperability remained free of all symptoms and carried on his work as a locomotive engineer for eight years. He then returned with an extensive recurrence of the neoplasm which again was removed, but it recurred and produced a fatal termination three years later. Fourteen patients were living and well when last heard from, from sixteen months to ten

TABLE 3.—*Summary of Results in Thirty-Eight Cases of Cancer of the Middle Ear and the Mastoid Process*

Date of Registration	Age (Yr.) and Sex	Site of Origin of Tumor	Pathologic Diagnosis	Treatment	Years of Freedom from Recurrence	Result
October 1919	54 M	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Repeated cautery excision and irradiation	Periods, 3½ to 5½	Died of recurrence 15 years after original treatment at clinic
April 1921	28 F	Intrinsic	Fibrosarcoma, grade 4	Mastoidectomy and irradiation; plastic correction of facial paralysis	8	Living and well more than 8 years
September 1922	71 M	Extrinsic	Basal cell epithelioma	Irradiation	..	Palliation; died of epithelioma May 1924
January 1923	57 M	Extrinsic	Squamous cell epithelioma, grade 3	Irradiation	..	Palliation; died of recurrence May 1927
March 1924	51 M	Intrinsic	Squamous cell epithelioma, grade 2	Irradiation	3	Receiving colloid gold at home 3½ years later
October 1924	43 F	Intrinsic	Squamous cell epithelioma	Exploration, mastoidectomy and irradiation	..	No benefit; died of abscess of temporal lobe 3 months later
January 1925	55 M	Extrinsic	Basal cell epithelioma	Radical mastoidectomy and removal	1	Probable recurrence 14 months later
March 1926	51 F	Intrinsic	Squamous cell epithelioma, grade 2	Exploration and irradiation	..	Probably palliation only; died (date unknown)
April 1928	50 M	Extrinsic	Adenocarcinoma (mixed tumor)	Excision May 1928 and April 1936 and irradiation	8	Returned with recurrence after 8 years; died of recurrence 3 years later
September 1928	70 M	Extrinsic	Adenocarcinoma, grade 3; squamous cell epithelioma, grade 3	Cautery excision, diathermy and irradiation	2	Well 2 years after treatment
January 1929	55 M	Intrinsic	Fibrosarcoma, grade 3	Biopsy and irradiation	..	Palliation; probably dead
August 1929	65 M	Extrinsic	Squamous cell epithelioma, grade 3	Cautery excision and diathermy	..	Died of meningitis 1 month after operation
December 1929	68 M	Extrinsic	Basal cell epithelioma	Diathermy and irradiation	2	Well 2 years after treatment
June 1930	54 M	Extrinsic	Adenocarcinoma (mixed tumor)	Excision and irradiation	8½	Died 10½ years after treatment (cause unknown)
October 1930	42 M	Intrinsic	Squamous cell epithelioma, grade 1	Exploration and irradiation	Unknown	Patient could not be traced
June 1931	50 F	Extrinsic	Adenocarcinoma and squamous cell epithelioma, grade 3	Diathermy, radical mastoidectomy and irradiation	10½	Well 10½ years
June 1931	45 F	Intrinsic	Hemangioendothelioma, grade 2	Radical mastoidectomy and irradiation	10	Well 10 years
March 1932	63 M	Extrinsic	Basal cell epithelioma	Diathermy, radical mastoidectomy and irradiation	9½	Well 9½ years
September 1932	54 F	Extrinsic	Basal cell and squamous cell epithelioma, grade 2	Diathermy	..	Sequestrum removed at home 4 months later; patient not traced thereafter

October 1932	52 M	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Repeated removal with cautery, diathermy and irradiation; dissection of lymph nodes	1+	Died of recurrence after 8 years
August 1933	47 F	Intrinsic	Squamous cell epithelioma, grade 4	Radical mastoidectomy and irradiation	..	Palliation; died of recurrence after 8 months
February 1934	59 M	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Diathermy and irradiation	7	Well 7 years
June 1934	50 F	Extrinsic	Basal cell epithelioma	Diathermy, radical mastoidectomy	7	Well 7 years
August 1936	43 F	Intrinsic	Squamous cell epithelioma, grade 3	Exploration of mastoid and irradiation	..	Palliation; died of recurrence after 16 months
November 1936	65 M	Extrinsic	Squamous cell epithelioma, grade 2	Cautery excision, diathermy and irradiation	..	Died of meningitis and septicemia 15th day
January 1937	44 M	Extrinsic	Squamous cell epithelioma, grade 1	Radical mastoidectomy and irradiation	..	Palliation; died 4 years later, probably of recurrence
February 1937	42 F	Intrinsic	Hemangioendothelioma, grade 1	Exploration, mastoidectomy and irradiation	3½	Well 3½ years later
October 1937	66 F	Intrinsic	Squamous cell epithelioma, grade 2	Biopsy and irradiation	3½	Well 3½ years
April 1938	59 F	Extrinsic	Basal cell and squamous cell epithelioma, grade 1	Diathermy, radical mastoidectomy and irradiation; dissection of lymph nodes	3¾	Well 3¾ years
April 1938	57 F	Intrinsic	Squamous cell epithelioma, grade 3	Radical mastoidectomy, diathermy and irradiation	⅝	Treated elsewhere for recurrence 8 months later
June 1938	48 F	Extrinsic	Adenocarcinoma (cylindroma)	Diathermy, radical mastoidectomy and irradiation	2½	Well 2½ years
December 1938	46 F	Intrinsic	Hemangioendothelioma, grade 1	Removal and irradiation	..	No recurrence after 4 years
October 1939	42 F	Intrinsic	Hemangioendothelioma, grade 1	Radical mastoidectomy and irradiation	2	Well 2¾ years
October 1939	80 F	Intrinsic	Squamous cell epithelioma, grade 2	Biopsy and irradiation	..	Probably palliation only; patient could not be traced
November 1939	27 M	Intrinsic	Hemangioendothelioma, grade 1	Exploration, mastoidectomy and irradiation	1⅓	No recurrence when patient was observed last 1⅓ years later
August 1941	37 M	Intrinsic	Hemangioendothelioma, grade 1	Exploration, mastoidectomy and irradiation	..	No recurrence after 9 months
September 1941	33 F	Intrinsic	Hemangioendothelioma, grade 1	Radical mastoidectomy and irradiation	..	No recurrence after 13 months
December 1941	3 F	Intrinsic	Neurofibrosarcoma, grade 2	Radical mastoidectomy, diathermy and irradiation	..	Died 3 months after operation

and a half years after treatment. In 3 cases operation was carried out less than six months prior to this report and accordingly the outcome is indeterminate.

SUMMARY AND CONCLUSIONS

Thirty-eight cases of cancer involving the middle ear and the mastoid process are reported.

This type of involvement occurs more frequently than the medical literature would lead one to believe, and the prognosis is more satisfactory than generally is thought.

Primary malignant (intrinsic) involvement of the middle ear and mastoid antrum by a neoplasm arising in one or the other of these situations occurs more frequently than does secondary involvement by a neoplasm originating in the pinna or in structures extrinsic to the ear.

The symptoms and other clinical features of intrinsic cancer at times are the same as those of inflammatory disease of the middle ear and the mastoid process, and exploration and biopsy may be necessary for diagnosis.

The most effective form of treatment for neoplasms of this type is a combination of electrocoagulation, radical mastoidectomy and irradiation, and in indicated cases this treatment often is of considerable benefit.

At the time of writing a number of the 38 patients in this series have been living and well for periods ranging from three to ten years after treatment.

INTRODUCTION TO THE p_H OF THE THROAT

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Although the throat is the site of the most common surgical operation in the world, it has been surprisingly free from that type of intensive physiologic investigation which, with some measure of success, has elevated nasal physiology into a position of well deserved prominence. As in the case of the nose, disturbances in the throat may be an expression of altered physiologic activity rather than the end result of gross pathologic changes. The more one examines the normal or the abnormal throat, the more firmly does this thought impress itself. The problems related to the physiology of the throat are many. The ground for painstaking investigation is rich and fertile. It requires only adequate ploughing.

PRESENT INVESTIGATION

The investigation I am about to describe can be characterized by a duality of approach: First, an effort has been made to determine the normal, physiologic range in the p_H present in the clinically normal throats of 51 men and women, and, second, the relationship of the p_H of the normal throat to the various varieties of local throat medication now available to the American public has been considered as a subject for future investigations. In his excellent book, "General Technic of Medication," Fantus [1] asserted that a hydrogen ion concentration near that which is normal for the mucous membrane is of even greater importance for applications to the mucous membranes than is isotonicity. While there are certain physiologic similarities between the p_H of the nose and that of the throat, sufficient differences arise to complicate the problem. Until these ramifications are studied no final suggestions can be advanced with regard to an appropriate p_H for throat medication.

Elsewhere I [2] have reported the use of a silver–silver chloride glass electrode in conjunction with the Coleman electrometer for determination of the nasal p_H.

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1. Fantus, B.: General Technic of Medication, ed. 3, Chicago, American Medical Association, 1938.

2. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ, Arch. Otolaryng. **34**:150 (July) 1941; Further Studies: Significance of the p_H of Nasal Secretions in Situ, ibid. **34**:297 (Aug.) 1941; Effect of Silver Preparations and Antiseptics on the p_H of Nasal Secretions in Situ, ibid. **34**:302 (Aug.) 1941.

The apparatus has again been harnessed, this time to the throat. When the patient is comfortably seated on a chair he is instructed to open his mouth widely. The glass electrode is inserted into the patient's mouth so that it is brought into contact with the mucous membrane of the supratonsillar region immediately adjacent to the upper pole of the tonsil. At this site the contact of a glass electrode can be tolerated by the patient; if, however, the electrode is placed on the postpharyngeal wall, the pharyngeal reflex immediately comes into play and interferes with the accuracy of the readings of the p_H. A tube containing a salt bridge makes contact with the patient by being placed on the opposite cheek, on the side opposite the glass electrode. The patient should remain as quiet as possible, for unnecessary movement at the time readings are taken will distort their accuracy. Readings are taken each minute for periods of one-half hour. Unlike the method utilized for the determination of the nasal p_H, the glass electrode cannot remain in situ in the throat for a long period. It is inserted; a reading of the p_H is taken, and the electrode is withdrawn after each reading. The futility of relying on one p_H reading only is amply illustrated by the frequent numerical changes in values that occur when readings are recorded for an appreciable period.

It will be recalled that the mucous membrane of the throat is lined by stratified squamous epithelium. Secretions from a number of sources—notably mixtures of the secretions of the parotid, submaxillary and sublingual glands, together with mucus secreted by the minute mucous glands scattered throughout the palate, cheeks, lips, tongue and gingiva—keep the mucous membrane of the throat moist and lubricated. In addition, there may be contact with secretions from the postnasal space and from the lower respiratory tract.

In any study of the p_H of the mucous membrane of the throat, it should be remembered that it is the total collection of the various secretions imposed on the membrane which influences the electrometric measurements. I have been unable to find in the medical literature any ready reference to the hydrogen ion concentrations of isolated secretions from the glands present in the palate, cheeks, lips, tongue and gingiva. There are, however, a number of papers on the p_H of saliva which throw partial light on the problem.

While the majority of older textbooks refer to mixed saliva—the product of all the salivary glands—as alkaline in reaction, a change is to be noted in more recent works, in which it is stated to be either slightly acid or neutral. The results of many recent investigations, particularly those of Brawley,[3] Starr,[4] Bunzell[5] and Cassarotto,[6] indicate that the average p_H of normal resting saliva (the term "resting saliva"

3. Brawley, R. E.: Studies of the p_H of Normal Resting Saliva, J. Dent. Research **15**:55, 1935.

4. Starr, H. E.: Studies of Human Mixed Saliva, J. Biol. Chem. **54**:43, 1922.

5. Bunzell, H. H.: Colgate Bull., 1922, nos. 1, 2, 3 and 4.

6. Cassarotto, G.: Richerche sulla reazione attuale della saliva umana, Stomatol. **25**:461, 1927.

being given to saliva obtained without any form of induced stimulation) lies on the acid side. Brawley [3] found the average p_H of normal resting saliva for 3,405 subjects to be 6.75. In 296 persons Starr discovered a variation in the p_H of saliva of 5.4 to 8.0, while in a similar number of subjects Bunzell found the p_H of the salivary secretion to vary from 5.3 to 7.2. Starr also maintained that emotional excitement changes the p_H of saliva. Broderick's [7] investigations showed variations in salivary p_H from 4.9 to 8.0. Bloomfield and Huck [8] revealed that freshly expectorated saliva from normal persons shows a p_H varying from 6.0 to 7.3, with the p_H of 80 per cent of the specimens falling between 6.6 and 7.1. The use of compound solution of sodium borate U. S. P. (Dobell's solution) caused an alteration in reaction for thirty minutes.

In their series of investigations on the p_H of saliva, Grossman and Brickman [9] demonstrated that the p_H level of saliva, i. e., the average p_H for each subject, varied in health and in disease. They found the range of the p_H of saliva to be from 5.0 to 8.0. The range of the p_H of saliva among persons in ill health was greater than among those in good health. Although the p_H level of saliva varied with different persons, it was fairly constant with the same person. The average diurnal p_H of saliva lay between 6.5 and 6.9. The nocturnal p_H was found to be lower than the diurnal p_H. There is a drop in p_H after eating, but it is sometimes preceded by a rise. In this connection it is interesting to compare broadly the relationship between saliva and nasal secretions in situ; elsewhere I have called attention to the fact that the nasal p_H varies with sleep and rest, with the ingestion of food and diurnally and nocturnally, and can be changed not only by infection but by alteration in a person's moods.

The p_H of the mucous membrane of the throat describes a series of high and low points. As in the case of the p_H of the nasal mucous membrane, at no time does it remain constant over an appreciable period. I have found the normal physiologic range of values for the p_H of the throat in 51 men and women with clinically normal throats to be from 4.9 to 8.0. For 54.8 per cent of the subjects the values were entirely within the acid range; for 7.8 per cent, entirely within the alkaline range, and for 37.4 per cent they fell within a slightly acid,

7. Broderick, F. W.: The Normal Reaction of Saliva, Am. Dent. Surgeon, January 1929, p. 11.

8. Bloomfield, A. L., and Huck, J. G.: The Fate of Bacteria Introduced into the Upper Air Passages: IV. The Reaction of the Saliva, Bull. Johns Hopkins Hosp. **31**:118, 1920.

9. Grossman, L. I., and Brickman, B. M.: Some Observations on the p_H of Saliva, J. Dent. Research **16**:409, 1937.

slightly alkaline range of fluctuations. From these observations it is obvious that the throat normally may be either acid or slightly alkaline. The claims advanced by some pharmaceutic manufacturers that their particular varieties of local throat medicaments "neutralize excess acidity in the mouth and throat" and that therefore they are superior products can indeed be challenged when it becomes clear that normally the p_H of the throat is, for the most part, within an acid range.

Various manufacturers of local throat medicaments with whom I have corresponded have indicated that their products, collectively thirty-six, vary greatly in p_H, from 2.5 to 11.6. That the differences in the p_H of these thirty-six products, used as sprays and gargles and with swabs, were the final result of a manufacturing process rather than of consideration of a physiologic p_H for throat medication soon became clear.

Before final suggestions can be made for an appropriate physiologic p_H for local throat medication, it will be necessary to determine, first, the range of p_H values for the throat in persons with disease of the throat—acute and chronic "sore throat," acute tonsillitis, acute pharyngitis, lymphoid hyperplasia and pharyngitis sicca, to mention a few important conditions—and, second, the effect of local throat medication on the p_H of the throat in health and in disease. These are some of the problems I hope to answer, the exigencies of the war permitting, before a final statement can be made as to appropriate p_H values for local throat medication.

SUMMARY AND CONCLUSIONS

The throat has been surprisingly free from that type of intensive physiologic investigation which, with some measure of success, has elevated nasal physiology into a position of well deserved prominence. The problems related to the physiology of the throat are many.

By conducting minute to minute readings of the p_H values for the mucous membrane of the throat, and utilizing a glass electrode and a Coleman electrometer, I have found the normal physiologic range of the p_H values for the throat in 51 men and women with clinically normal throats to be from 4.9 to 8.0. For 54.8 per cent of the subjects the values were entirely within the acid range; for 7.8 per cent, entirely within the alkaline range, and for 37.4 per cent they fell within a slightly acid, slightly alkaline range of fluctuations. From these observations it is clear that the throat normally can be either acid or slightly alkaline.

The claims advanced by some pharmaceutic manufacturers that their particular varieties of local throat medicaments "neutralize excess acidity in the mouth and throat" and that therefore they are superior products

can indeed be challenged when it becomes clear that normally the p_H of the throat is, for the most part, within an acid range.

Before final suggestions can be made for an appropriate physiologic p_H for local throat medication, it will be necessary, in the course of future investigations, to determine, first, the range of p_H values for the throat in persons with disease of the throat and, second, the effect of local throat medication on the p_H of the throat in health and in disease.

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THE FENESTRATION OPERATION FOR OTOSCLEROSIS

REPORT OF THIRTY-TWO CASES

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When an account of the one stage endaural fenestration of the labyrinth for the relief of otosclerosis was first published, I refused to attempt the operation because I believed there was a definite danger of a meningitis due to the opening of the internal ear which the technic required. After being convinced there was no undue danger from this source, I decided to learn from personal experience the merits of this work.

Since July 1940 I have performed the operation in 32 cases. In the first 18 of these cases I followed Lempert's [1] original method, which involved exenteration of the mastoid, removal of the head of the malleus and fenestration of the horizontal semicircular canal with the incus in its normal position. In the remaining 14 cases I used the Lempert [1a] "fenestra nov-ovalis" technic, or what I shall call his "new method." With this new method the operator, after exenterating the mastoid, removes the incus and the head of the malleus and creates the fistula over the dome of the vestibule. A number of good results were obtained with the original method, and the proportion of successful operations has been materially increased by the new fenestra technic. By a good result I mean the restoration of hearing for the normal speech range. Unless the patient regains hearing which is useful in everyday contacts, I am inclined to regard the results as negative, a gain of 10, 20 or 30 decibels being of no value to the patient unless the gain reaches the practical threshold of ordinary conversation. Hearing was improved immediately when the labyrinth was opened, but in the cases in which the result was unsuccessful the decline was gradual from the sixth to the fourteenth week, a longer time being required in some cases before failure became apparent. The longest period in which the improvement has been maintained is now over two years, another is twenty-two months and another eighteen months. Generally speaking, if the improvement is retained for eight months one feels, from present

1. Lempert, J.: Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **28**:42 (July) 1938.

1a. Lempert, J.: Fenestra Nov-Ovalis, Arch. Otolaryng. **34**:880 (Nov.) 1941.

knowledge, that it will be lasting; more time and experience are necessary to be sure that it will be permanent.

In 3 of the first 18 cases a second operation was carried out, and in all 3 instances the fistula was observed to be covered with solid new bone. Hearing was again restored after removal of the new bone over the fistula, but in 2 of these cases it again receded, undoubtedly owing to closure of the bone. In the third case the fistula has remained open, and the improvement in hearing has been maintained. It is important to note that all three revisions were done without any ill effects or complications; however, great care must be taken to avoid injuring the membranous labyrinth when one is removing the secondary layer of bone and the endosteum.

In my first 18 cases it was discouraging to have the fistula close in so many instances, but when it is remembered that prior to the use of the Lempert technic nothing had ever been accomplished in cases of otosclerosis, the results were encouraging. With the use of the new method, the results have been even better. As Lempert has shown, the fistula, when made farther forward, over the dome of the vestibule, can be made wider and is created through a thinner layer of bone, while the removal of the incus allows Schrapnell's membrane and the thin integument immediately adjoining it to be placed over the fistula. This was not possible, in my opinion, with the incus in its normal position. These factors have undoubtedly increased the chances of success in this work.

Considering the question as to when the operation is indicated, one must keep clearly in mind that this procedure is essentially an operation for conductive deafness. If there is sufficient nerve degeneration or true nerve deafness, the operation is absolutely contraindicated. This means that the bone conduction should be above the 25, and preferably above the 20, decibel level for 512, 1024 and 2048 frequencies. The drum membrane should be intact and the eustachian tube patent, and there should be no previous infection of the middle ear. Even with the history of such an infection, the operation may be undertaken if the drum has healed perfectly and sufficient time has elapsed since the infection occurred.

The diagnosis of otosclerosis seems best to fit this picture. On the other hand, there seems to be no way to differentiate between true otosclerosis and some of the so-called advanced stages of catarrhal deafness. In any event, when an operation is considered, the primary requisite is that there be sufficient bone conduction present to enable the nerve to transmit the sounds after the fistula is established.

The audiograms made in 11 cases accompany this report.

In case 1 (fig. 1 *A*) the first operation was performed on July 25, 1940, and practical hearing was restored, but after three months it gradually receded to its

original status. On June 16, 1941 the second operation was done, and the fistula was observed to be closed with new bone; after removal of the bone the hearing returned and has been retained. It is now more than one year and two months since the operation.

In case 2 (fig. 1 *B*) there is a very active fistula, which can be demonstrated by pressure on the external ear. There is satisfactory practical hearing, with a gain of 23 decibels, now more than one year and nine months after operation.

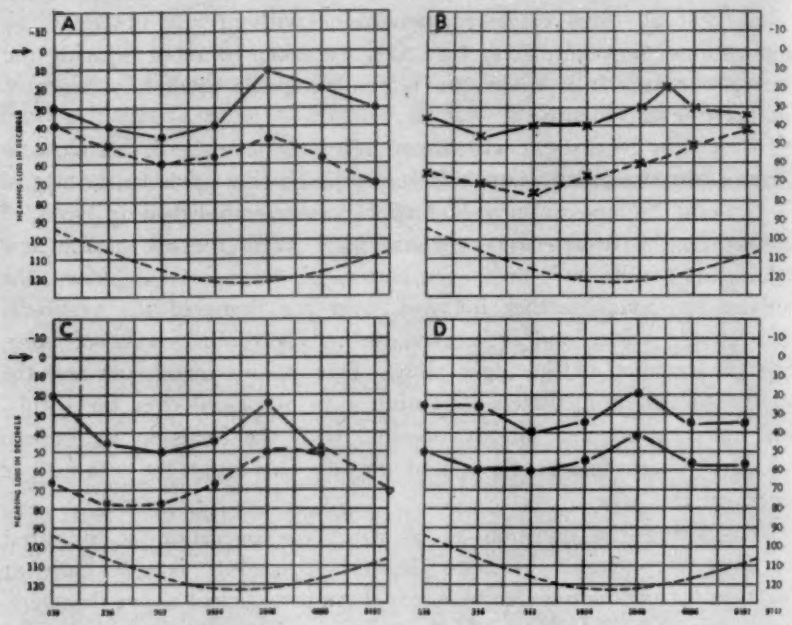

Fig. 1.—*A* (E. W., a woman aged 24): preoperative hearing curve and original fenestration operation on the right ear, July 25, 1940; revision June 16, 1941; postoperative hearing curve, Aug. 18, 1942, one year and two months after the revision and two years and one month after the original operation; hearing gain 23 decibels.

B (B. W.): preoperative hearing curve and original fenestration operation on the left ear, Sept. 6, 1940; postoperative hearing curve, June 29, 1942, one year and nine months after operation; hearing gain 23 decibels.

C (P. C., a man aged 49): preoperative hearing curve and original fenestration operation on the right ear, Nov. 24, 1940; postoperative hearing curve, Oct. 5, 1942, one year and eleven months after operation; hearing gain 17 decibels.

In this figure, and in the accompanying figures, average normal hearing for ages from 18 to 23 is indicated by the arrow. The preoperative hearing curve is shown by a broken line, and the postoperative curve, by a solid line. Air conduction for the right ear is indicated by a line with circles; that for the left ear, by a line with crosses.

D (M. H., a woman aged 38): preoperative hearing curve and original fenestration operation on the right ear, March 3, 1941; postoperative hearing curve, Aug. 7, 1942, one and a half years after operation; hearing gain 16 decibels.

In case 3 (fig. 1 *C*) decided improvement followed operation; then a pronounced recession occurred, with a small perforation in Schrapnell's area. After this closed, the hearing again improved, and there is a good practical result, with a gain of 17 decibels, which is maintained one year and eleven months after operation. The patient is now 51 years of age.

In case 4 (fig. 1 *D*) an active fistula, with good practical hearing, exists more than one and one-half years after operation.

In case 5 (fig. 2 *A*) hearing showed good gain, but the acuity present the first few weeks after operation was not retained. The postoperative period is one year and four months.

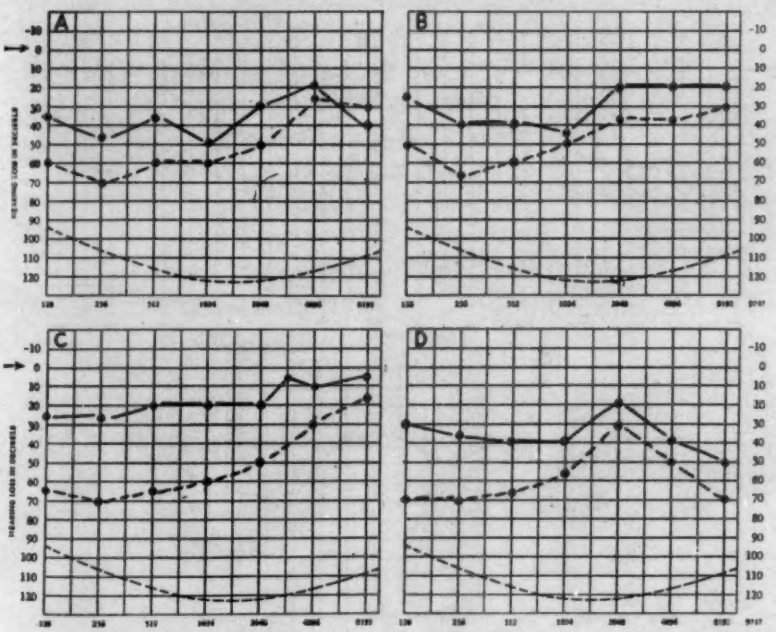

Fig. 2.—*A* (M. C., a woman aged 30): postoperative hearing curve and original fenestration operation on the right ear, March 27, 1941; postoperative hearing curve, Aug. 11, 1942, one year and four months after operation; hearing gain 15 decibels.

B (S. N., a woman aged 25): preoperative hearing curve and nov-ovalis fenestration operation on the right ear, Nov. 13, 1941; postoperative hearing curve, Aug. 3, 1942, nine and a half months after operation; hearing gain 13 decibels.

C (J. L., a girl aged 16 years): preoperative hearing curve and fenestra nov-ovalis operation on the right ear, Jan. 28, 1942; postoperative hearing curve, Aug. 18, 1942, eight months after operation; hearing gain 30 decibels.

D (P. C., a girl aged 14): preoperative hearing curve and fenestra nov-ovalis operation on the right ear, Feb. 2, 1942; postoperative hearing curve, Aug. 3, 1942, six months after operation; hearing gain 14 decibels.

In case 6 (fig. 2 *B*) an active fistula exists. The gain in hearing for the higher frequencies seems to be satisfactory from the standpoint of practical conversation.

This case represents the first successful result with the so-called new method, or fenestra nov-ovalis operation, and is the second case in which the new technic was used. In the first case in which the new method was employed hearing receded, apparently as a result of closure of bone. To date, I have not done revision in any of the cases in which I have used the new technic.

In case 7 (fig. 2 *C*) the result is especially fine. It should be noted that the patient was only 16 years of age. Her hearing was so poor that she was preparing to abandon school and study lip reading. This patient has a very active fistula, which can be seen with the naked eye. There was an improvement in hearing of 30 decibels, which is still maintained, more than nine months after operation.

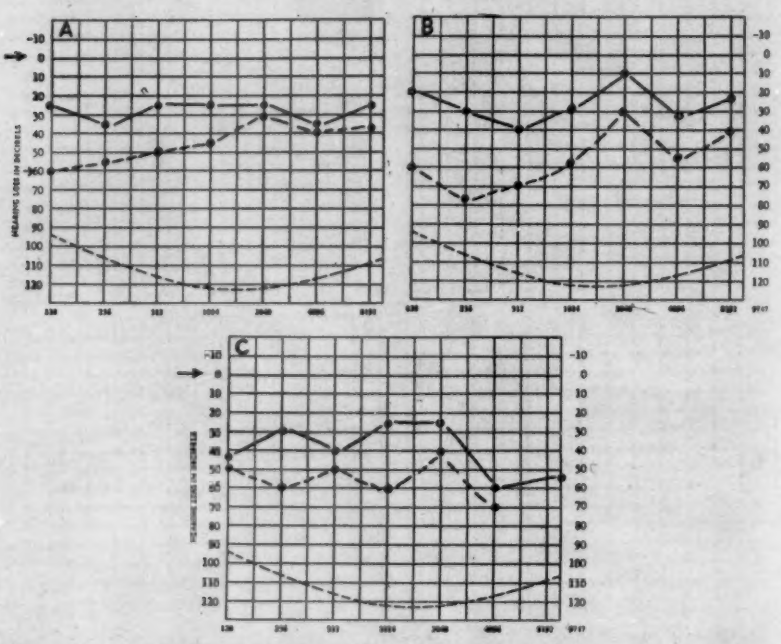

Fig. 3.—*A* (J. S., a girl aged 16): preoperative hearing curve and fenestra nov-ovalis operation on the right ear, March 14, 1942; postoperative hearing curve, Aug. 3, 1942, five and a half months after operation; hearing gain 19 decibels.

B (H. D., a woman aged 25): preoperative hearing curve and fenestra nov-ovalis operation on the right ear, June 10, 1942; postoperative hearing curve, Aug. 5, 1942, three months after operation; hearing gain 20 decibels.

C (A. A., a man aged 48): preoperative hearing curve and fenestra nov-ovalis operation, May 13, 1942; postoperative hearing curve, Aug 3, 1942, three months after operation; hearing gain 16 decibels.

In case 8 (fig. 2 *D*) the patient was only 14 years of age. There is an active fistula, with very good hearing.

In case 9 (fig. 3 *A*), also that of a girl 16 years old, hearing shows improvement, which should be retained.

In both cases 10 and 11 (figs. 3 *B* and *C*) the result is good, but more time must elapse before I can be sure that the gain will be permanent.

It will be noted that the postoperative hearing tests on these audiograms were made in August 1942, and more than six months has elapsed between that time and the publication of this article.

The hearing has been maintained to February 1943 in all of them, so that an additional six months may be added to their postoperative duration.

When one studies the audiograms as a whole, one notes that most of them show good improvement in the area of the 2048 frequency, which seems to produce satisfactory practical hearing despite the lack of any notable improvement in the 512 and 1024 frequencies in some instances.

The oldest patient operated on was 49 and the youngest 14 years of age. In my opinion, good results should be secured with the older, as well as the younger, patients if the bone conduction is satisfactory. provided of course that extreme age does not make the subject a poor operative risk. In some of the other cases in which the fenestra novovalis technic was used good results were obtained, but the postoperative period has not been long enough for them to be included in this report.

All the patients were operated on at the New York Eye and Ear Infirmary. The audiograms were made by the house officers, and the cases are all on record at this institution.

Unfortunately, a soundproof room was not available for the hearing tests in these cases. Dr. E. P. Fowler made the final postoperative audiograms in cases 2 and 7 in a soundproof room, and in both instances they showed more improvement than the audiograms made in the hospital.

COMMENT

There is a difference of opinion among the men doing this work regarding the merits of the endaural and the postauricular approach to the operative field. I prefer to do the simple and the radical mastoidectomy by the postauricular route. I performed the first fenestration operation through a postauricular incision, but all the subsequent ones have been done by the endaural method. This is the best approach for the fenestration operation in my opinion, because of the facility with which the flap can be constructed and bleeding controlled, two important factors in this work.

From a practical viewpoint this work is in its infancy. There are many problems which future records and investigations should solve or help to clarify. Some of them may be stated as follows:

Is the nerve degeneration in some of the cases due to stagnation or atrophy of the nerve endings from nonuse or to lack of stimulation as

a result of the sound impulses to reach the inner ear because of the fixation of the stapes?

Will an early operation prevent this degeneration, and, if so, will it be permanent?

What results can be obtained with good bone conduction for the 512 and 1024 frequencies but a drop for the 2048 frequency? How low should the recession be for the 2048 frequency before the operation is contraindicated?

There is a difference in the tissue reactions of different persons. In one case the drum flap and other tissues will undergo a conspicuous inflammatory reaction, and in another the reaction will be mild, the normal color of the drum membrane returning quickly. In another case a marked secondary infection will be present in the tissue covering the roof and the back of the mastoid cavity and will take a long time to clear up regardless of the after-treatment, while in still another the ear will become dry a short time after the operation. It seems that a severe secondary reaction would be more detrimental to the end result, but to date there is no definite proof of this unless a true labyrinthitis should result.

There is also the problem of establishing a much better preoperative classification and diagnosis, which should materially lower the number of failures and make possible a more accurate prognosis in the future.

What part does the blood which accumulates in the field of the fistula, at the time of the operation, play in the closure of the fistula? In some of the cases it is possible to work and to create the fistula in a practically bloodless field; in others it is almost impossible. Nevertheless, some of the most successful results have occurred in cases in which the bleeding has been most troublesome. It is especially necessary to have the field of the fistula as dry as possible immediately before it is finally covered with the flap.

What part does fibrosis instead of osteogenesis play in the retrogression of the hearing in some of these cases?

Finally, there is the main problem of bone regeneration. Why of 2 cases in which operation is performed in exactly the same way will there be a closure in one case and a good result in the other?

Despite past experiments and the work now being done, a complete solution of this problem does not seem possible in the very near future. There is every reason to hope that continued research will throw some light on this subject and result in an increase in the number of successful results.

The work done in the cases reported in this article confirms the claim of Lempert that with proper asepsis and technic this surgical procedure can be performed without undue risk, and therefore the results

justify in every way the continuation of this research. There was no facial paralysis, meningitis, intracranial complication or diffuse labyrinthitis—only the labyrinthine disturbance due to the opening of the internal ear, which quickly subsided. This means that the hearing in the cases in which the fistula closed was not made worse but merely receded to its preoperative level. These results were made possible by proper learning of the technic through many hours of hard work on the cadaver before the operation was attempted and by the full cooperation of Dr. Lempert, who unhesitatingly helped in every way with his past experiences in this work.

When it is remembered that the hearing of all the patients was below the practical threshold, there was little to lose and everything to gain by the operation.

Sporadic attempts to restore the hearing in cases of otosclerosis by creating an opening in some part of the labyrinth capsule have been made at different times as far back as the beginning of the eighteenth century. To Holmgren,[2] of Sweden, and Jenkins, of England, must go credit for stimulating a practical interest in this problem, dating from 1914, and to Sourdille,[3] of France, both for reviving this interest when Holmgren admittedly was ready to give up because of his many failures and for devising his tympanolabyrinthopexy, or three stage technic, which has contributed so much to the individual steps of the present operation. Then to Lempert great credit is due for his unceasing toil and effort, resulting in a magnificent technic which makes possible the practical restoration of hearing by a one stage operation.

Since it is not possible to guarantee results in any case, it is my custom to explain the possibilities and probabilities to the individual patient and to let him decide whether or not he wishes the operation.

Any one who has had the privilege of examining before and after operation some of the patients for whom successful results have been obtained, especially the 3 children whose audiograms are shown in figures 7, 8 and 9, would readily be convinced of the importance and merit of this work. In my opinion, this is an exceedingly important type of research, one to which otologists should give every possible encouragement. There will be many failures, owing to the complex nature of the procedure; for this reason, the operation will have difficulty in assuming its proper place in surgical otology. It is necessary to emphasize the fact that there is definite danger of facial paralysis,

2. Holmgren, G. J.: The Surgery of Otosclerosis, Ann. Otol., Rhin. & Laryng. **46**:3 (March) 1937.

3. Sourdille, M.: New Technique in the Surgical Treatment of Severe and Progressive Deafness from Otosclerosis, Bull. New York Acad. Med. **13**:673 (Dec.) 1937.

diffuse labyrinthitis and meningitis when the work is in the hands of the untrained technician. The operation, therefore, should be attempted only by the otologist who is thoroughly trained and experienced in mastoid surgery. It must first be taught on the cadaver, and then the necessary time and effort must be devoted to the correlation of the intricate steps comprising the technic before it is carried out on the living. It will not be possible to operate on an occasional patient with satisfactory results; therefore, this research will perhaps best be carried out in the large universities and clinics, where suitable material, together with the proper facilities for recording results, are available.

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MALFORMATION INVOLVING EXTERNAL, MIDDLE AND INTERNAL EAR, WITH OTOSCLEROTIC FOCUS

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BOSTON

A deformed auricle and an occluded external auditory canal are indications that there may be further malformations within the confines of the middle and the internal ear. Such a situation occurred in the following case.

REPORT OF A CASE

A white girl aged 10 years was struck by an automobile and died in the hospital after a few hours. The body was 59 inches (150 cm.) long; multiple fractures of the base of the skull were found. The left ear was of normal size and shape, but on the right side there was only a cartilaginous ridge, 3 cm. long, crescentic in shape and concave anteriorly. In the center of the concavity a small orifice admitted only the head of a probe. Three horizontal grooves, 3 to 5 mm. in depth, were present above the ridge. The petrous pyramid was narrower on the affected side, and its transverse measurement across the intracranial surface over the tegmen tympani was 12 mm., as compared with 16 mm. on the normal side.

Relatives stated that the hearing was normal in the left ear and that no hearing had been present on the right side since birth.

The affected temporal bone was removed and was examined roentgenologically, and the roentgenograms were interpreted by Dr. A. S. Macmillan, who gave the following report: "The petrous apex is cancellous, with suture lines showing between it and the surrounding bone. The cochlea and semicircular canals are fully developed. The lateral sinus, jugular bulb and mastoid process can be recognized, but there are no pneumatic cell spaces. The ossicles and external canal cannot be made out. The internal auditory meatus appears to be normal."

After the specimen was decalcified and embedded in celloidin (pyroxylin), four hundred horizontal sections were cut. Microscopic examination revealed extensive malformations in all three main portions of the ear.

The mastoid process, of brachycephalic type, showed no pneumatization. The bony trabeculae were of the normal lamellar type, with numerous rows of osteoblasts at their edges. The cancellous bone presented only marrow spaces, except that some of the spaces in the center and along the anterior wall were filled with solid fibrous tissue instead of marrow. These spaces also showed extensive osteoblastic activity resulting in osteoid margins. The anterior and posterior walls of the mastoid were formed by solid cortical bone. The mastoid was traversed by

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a closed tubular cavity nearer the anterior wall, running in a mediolateral direction; the walls of this tube were of cortical bone. The lumen was biscuit shaped, with a somewhat bent axis. Medially a solid bony plate separated it from the tympanic cavity. The medial part of the tube was filled with solid fibrous tissue with a poor blood supply. Laterally the cavity was hollow except for an exostosis in the external half. It arose by a broad pedicle from the posterior wall and had an irregular shape and dense lamellar structure, with only a few connective tissue spaces. In the lower sections, in which the entire cavity was filled by connective tissue, a narrow fissure could be traced almost through the whole length of the fibrous plug. This fissure was lined by the aforementioned periosteum and epithelium. The cavity could be followed from the first sections

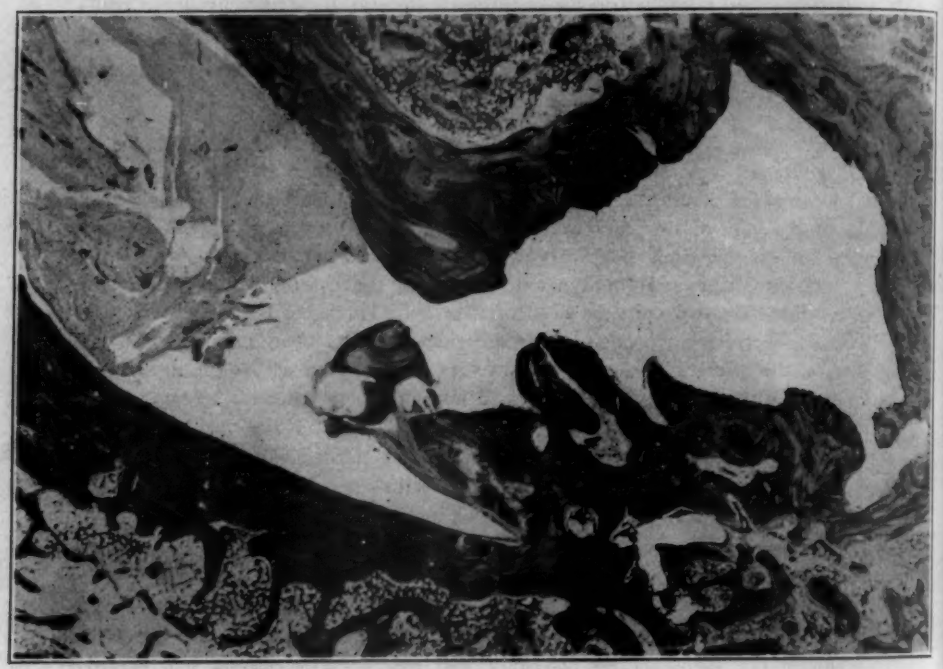

Fig. 1.—Cavity running across the mastoid, with a fibrous plug in the medial portion and exostosis in the lateral half.

down to section 170; it was closest to the tympanic cavity about in section 80. Even at this area there was a bony septum separating it from the tympanic cavity; it also jutted into the tympanic space.

Anterosuperior to the mastoid there was a well defined pea-sized space corresponding to the antrum. It was separated incompletely from the tympanic cavity by a bony septum protruding from the lateral wall. Between this space and the mastoid cells no connection was found. Inferiorly another opening was found between this space and the tympanic cavity.

The tympanic cavity was seen in all the sections. Its posterior wall contained some larger cells filled with marrow; the anterior wall narrowed down to a single row of cells. The epitympanum was filled with dense connective tissue;

the lower part of the tympanic cavity was open in the tubal region. The contained connective tissue had small, straight vessels; in scattered areas vacuoles appeared. The mucous membrane did not follow the bony wall and did not line the vacuoles; it covered only the tubal ostium and the adjacent part of the tympanic wall, going over to the free edge of the pad of connective tissue. It was of simple squamous type, with well stained nuclei. There was a small hemorrhage, covering a part of the mucosa. Near the tubal ostium the epithelium seemed to be cuboidal.

There were only two ossicles, one of them the stapes. The first ossicle began at the roof of the epitympanum; in cross section it was triangular at the top,

Fig. 2.—The cavity in the mastoid is compressed to a narrow fissure amid cancellous bone. At the site of the membrana tympani a bony plate closes off the tympanic cavity.

elliptic in the mesotympanum and cylindric at the hypotympanum. A marrow space lay in the center. The bony external surface was penetrated at several points by connective tissue which joined the central marrow space. These penetrating channels were lined with osteoid tissue. The attachments of this ossicle were varied. In the epitympanum several ligaments were present, which were formed by condensations of the connective tissue. Between sections 150 and 170 the ossicle gained attachment to the lateral wall of the tympanic cavity through a short, bony bridge. Below this bridge it formed a part of the wall of the canal for the facial nerve. It lay between the facial nerve and the stapedius

muscle. At a lower level it formed still a greater portion of the wall of the canal for the facial nerve and continued downward toward the stylomastoid foramen.

The stapes was very abnormal. Its plate was in its normal locus, but the base of the lateral crus reached only to the center of the footplate, falling short of its normal location. In this way the lateral half of the plate presented only a simple bony septum. There was no capitulum, the crura converging in a smoothly rounded dome. The lumen was completely filled with connective tissue, in the center of which there was an oval, sharply circumscribed collection of round cells. The annular ligament was partly replaced by bone at its anterior and posterior margins.

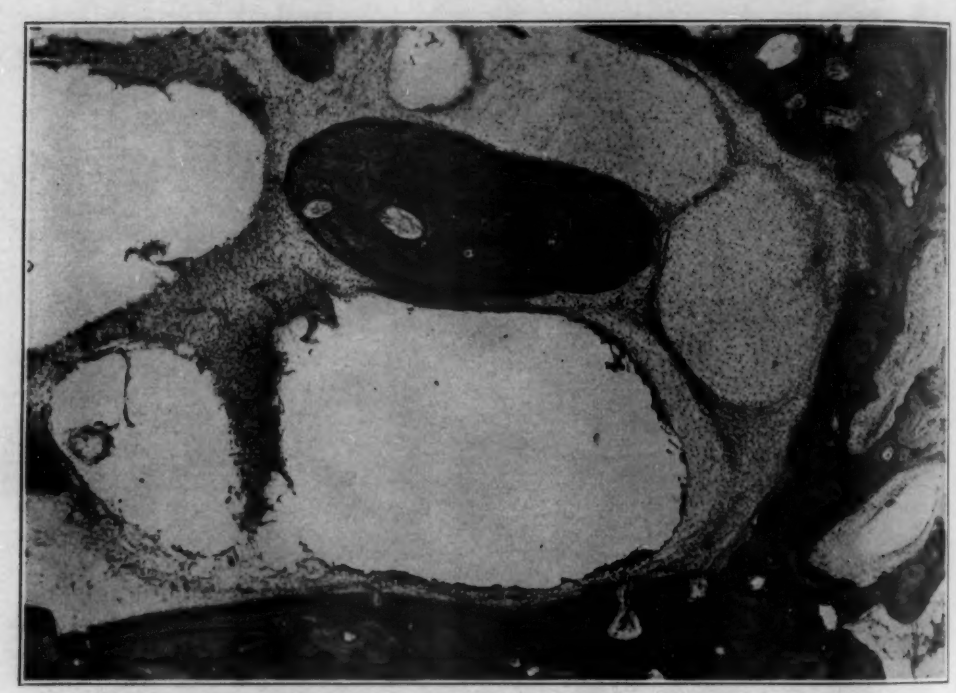

Fig. 3.—A single bone replaces the malleus and incus in the epitympanum, with ligamentous attachments.

This replacing bone was like the surrounding bone, but near the lateral border of the footplate there was a small focus of reticulation representing the earliest stage of otosclerotic change.

The round window was open, but the niche and the adjacent parts of the tympanic cavity were filled by a solid pad of connective tissue and the secondary tympanic membrane bulged into the tympanic cavity.

The facial nerve could be followed in its entire length and showed no dehiscence in the wall of the canal. The cells of the geniculate ganglion were well developed.

The stapedius muscle was of normal size and structure, but the tendon was absent. The muscle ended blindly in its conical cavity within the pyramidal eminence.

The tensor tympani muscle was replaced by a fibrous band without striations. No tendon was present.

The tubal ostium was wide open, with some fibrinous masses along its wall. Remnants of connective tissue within the lumen contained conglomerate small vessels, indicating resorption.

The inner ear showed normal development of the bony capsule, but there were malformations of the central bony structures in the cochlea.

The vestibular labyrinth was of normal architecture, but the maculas and cristae were considerably flattened, with their nerves atrophic, leaving many of the respective canals for their filaments empty.

Fig. 4.—The same bone shown in figure 3 in the mesotympanum, with osseous fixation to the lateral wall of the tympanic cavity. The tympanic cavity is partly filled with solid connective tissue and partly open, the patent part lined with mucous membrane.

The capsule of the cochlea was normal in shape and structure, but the modiolus did not reach the apex. It was of normal size at its base but reached only to the middle of the second turn. Here the spiral canal thinned out to the width of the osseous spiral lamina. Near the cupula it spread out to form the two larger ducts of the apical turn. In the basal turn, near the round window, there was an opening in the wall, at the site of the internal orifice of the cochlear aqueduct, but it seemed to end blindly. Corti's organ in all the three turns was a flat mound of dense undifferentiated tissue. The shortened modiolus, the lamina spiralis and the basilar membrane were present, with all the turns formed, but the scala vestibuli was bulged outward in the lower and middle turn. Since

there was complete absence of Reissner's membrane, the ductus cochlearis was missing. The stria vascularis was flattened in its whole course. There were nerve fibers between the upper and lower lamellas of the lamina spiralis ossea, but many of the bony spaces of the ganglion spirale were empty and the main trunk of the cochlear nerve in the central canal was atrophic. Similarly, the vestibular cochlear branches were atrophic in a widened internal meatus, which did not present the funnel-shaped antechamber (fig. 9).

Only a few hemorrhages were found, mostly in and about the tympanic cavity. The only region where the extravasation was of possible clinical significance was about the vestibular nerve in the internal acoustic meatus.

Fig. 5.—The same bone shown in figures 2 and 3, forming part of the canal for the facial nerve, the latter directly abutting the stylomastoid foramen.

Several microscopic fissures were found, e. g., between the vestibule and the posterior ampulla. They were filled with a tissue partly of a transparent homogeneous nature and partly fibrous.

COMMENT

Marx,[1] summarizing the reports regarding malformations of the middle ear, found 54 cases in the literature up to 1926. He emphasized that an exact microscopic examination with the use of serial sections

1. Marx, H.: Die Missbildungen des Ohres, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, pp. 609-734.

is the only way to arrive at an accurate understanding of the disposition of the soft tissues and the more intimate structure of the atresia. In recent years only a few cases have been studied in this manner.

This right-sided anomaly fits into the greater frequency of right-sided aural anomalies as studied by Hrdlička [2] in a number of skulls of pre-Columbian origin and also fits into the clinical observations of Beck.[3] The latter cited S. Michel's explanation: that the right sidedness of most anomalies is in relation to the position of the head in utero.

Fig. 6.—Deformed stapes, showing the crura only above the medial half of the footplate. The space between the crura is filled with connective tissue. There is no capitulum. The rudimentary stapedius muscle is without a tendon.

Three other right ears, unreported, with deformities of the auricle and atresia of the external meatus, have also come under my observation.

There are according to Marx two main groups of such anomalies, one with (16 per cent) and the other without (74 per cent) a tympanic membrane. The latter group is subdivided in three subgroups on the

2. Hrdlička, A.: Seven Prehistoric American Skulls with Complete Absence of External Auditory Meatus, Am. J. Phys. Anthropol. **17**:355-377 (Jan.-March) 1933.

3. Beck, J.: The Anatomy, Psychology, Diagnosis and Treatment of Congenital Malformations and Absence of the Ear, Laryngoscope **35**:814-831 (Nov.) 1925.

basis of changes in the tympanic cavity. Group 3 would indicate complete absence of this cavity. The anomaly in my case has to be placed between group 1, in which the size of the tympanic cavity is normal or at least not constricted but the ossicles are partly misshaped, and group 2, wherein there is partial obliteration of the tympanic cavity by osseous tissue and absence of advanced malformation of the ossicles. It does not fit exactly into any of the given groups and in more than one detail differs from all the hitherto reported cases.

Fig. 7.—Osseous fixation in the annular ligament and cartilaginous foci in the labyrinthine capsule.

The malformation of the auricle is one of grade 3, according to the classification of Marx, and fits into a very early period of development. This period corresponds to the first weeks of embryonal life, because at the end of the first month there is marked differentiation. The three small horizontal grooves above the rudimentary auricle represent impeded closure of the fetal fissure of the cheek, being located anterior to the auricle. They demonstrate a faulty blending of the tubercles around the margins of the first branchial arch.

The canal replacing the external meatus but running through the mastoid process is a unique feature. The osseous atresia is sometimes merely an incomplete fissure in compact bone, which may widen in its external or internal half. Here a plate is well developed against the

tympanic cavity, but no such situation occurs at the external surface, where the wall is formed simply by the cells adjoining the cortex of the mastoid process.

The question as to how the canal came to run along the horizontal central axis of the mastoid can be answered in several ways. The normal meatus is abutted by the cells of the mastoid only at its posterior and superior walls. This particular canal may be formed as an invagination from the external surface (as normally) or from the tympanic cavity outward. There are two features which support the possibility that the

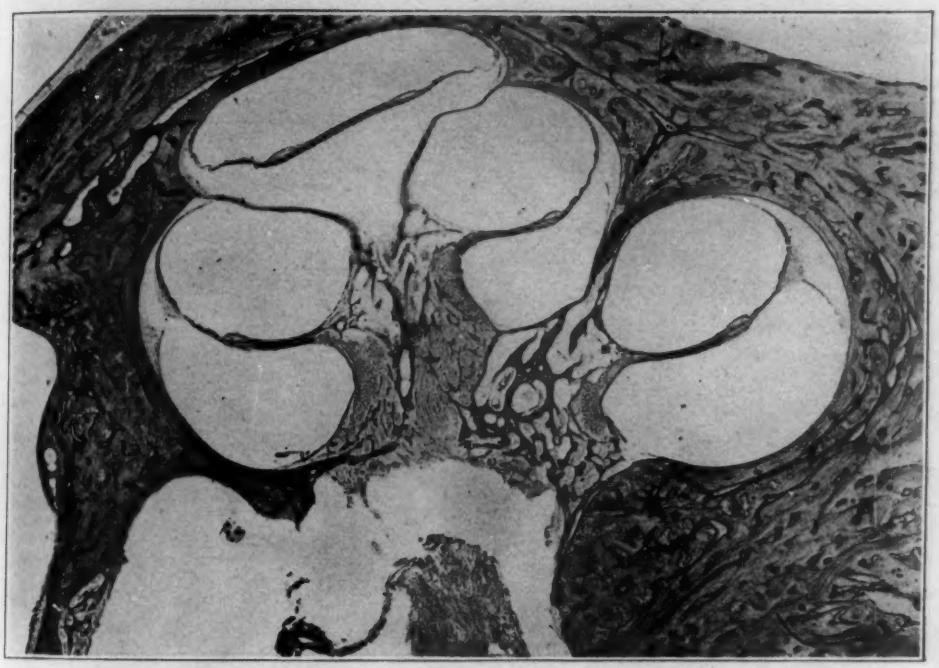

Fig. 8.—Well formed cochlear capsule. The modiolus reaches only the middle turn, and many of its spaces are empty. The scala vestibuli is distended, and there is no cochlear duct. Corti's organ is a low mound.

beginning was, as usual, the canalization of the ectodermal cord: first, the lining of the open part of the cavity with epithelium approaching the squamous-stratified type and, second, the exostosis, a typical meatal structure. After the development of the internal occluding plate from the tympanohyal, the canalization of the external plate of the latter occurred in a faulty, deviated direction, and the mastoid process took part in this abnormal development later, its cells secondarily surrounding the deviated canal. The sequence of the development thus may likely be: (1) the internal bony plate, (2) the rudimentary canal and (3) the mastoid process forming the lateral occlusion.

There is a possibility, as suggested to me by Dr. M. H. Lurie, that this cavity may have been formed by an ingrowth of epithelium from the tubal orifice, as an attempted pneumatizing process. A secondary occlusion by a bony wall may have formed later. The presence of the epithelium in the canal could be explained in this way also, but not so easily the presence of the exostosis. This exostosis reminds one of cases in which at operation on the mastoid process cavities were found containing attached or loose osseous inclusion bodies (Germán [3a] and others). How-

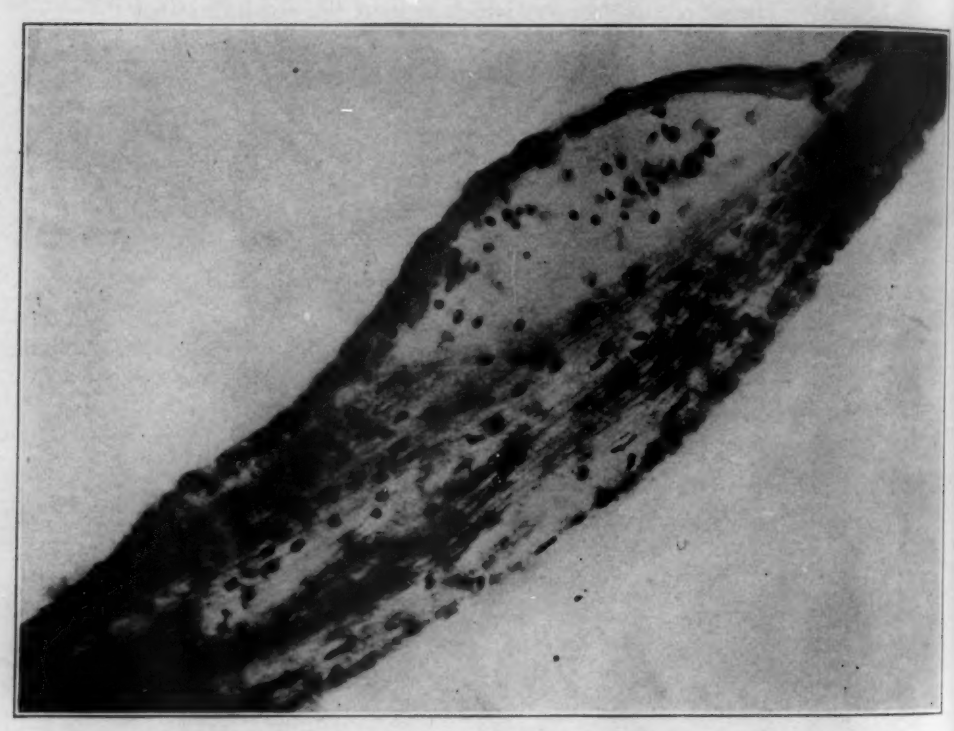

Fig. 9.—Corti's organ, a flat solid mass of undifferentiated cells.

ever, the latter originated from inflammatory processes, and no signs of such changes are to be found in this mastoid. The exostosis with its development from a rest with a strong tendency to exuberant ossification would occur more readily in a rudimentary meatus than in a pneumatic cavity.

In contrast to this case, in which the duct in the direction of the cavum tympani was displaced backward, there are cases in which a fistula reaches the middle ear from an orifice in front of the ear, originating behind the angle of the jaw. Marx found reports of 2 such cases (by Riedel and De Ruyter).

3a. Germán, T.: Ueber die Entstehung multipler Knocheneinschlüsse im Warzenfortsatz, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:339-349 (June) 1927.

Some tracts of the fibrous tissue in osseous cells of the respective locations may represent replacements of the chorda tympani, but it is impossible to decide definitely. The connection between the chorda tympani and the occlusion plate makes it often take part in abnormal formations, and frequently its absence is reported. The time of formation of the bony plate, closing laterally the tympanic cavity, is not easily determined; its lower part, being in connection with the styloid process, forms earliest and the higher part, around the antrum, in a much later period of fetal life. A clue is given in this case because of the absence

Fig. 10.—Young otosclerotic focus near the annular ligament, with blue mantles about the vessels, cartilaginous islands and reticulation.

of the chorda tympani. The latter appears at a period previous to the primordium of the tympanic bone. The plate—at least in this case—represents an abnormal proliferation in the structures of the second (hyoid) arch, a purely branchiogenic formation.

Following the description of Richards,[4] from the "laterohyal" cartilage, "which should normally produce the styloid process, there arises a laterally extending bony outgrowth which enlarges to form an occluding mass of bone in the region of the drum membrane and annulus."

4. Richards, L.: Congenital Atresia of the External Auditory Meatus, Ann. Otol., Rhin. & Laryng. **42**:692-711 (Sept.) 1933.

The facial nerve was well developed in its course through the ear, but Jacobson's nerve was not found. The contrary finding, namely, absence of the facial nerve and a normal Jacobson nerve, was mentioned by Cantele [5] in his case report, so far unique, with histologic description of a malformation of the ear in connection with congenital hydrocephalus.

In the tympanic cavity, which was of normal size, it is remarkable that the mucous membrane was not formed between the bony wall and the soft tissues but developed only as a lining of the free cavities, in the tympanic cavity itself covering the open space originating from the tube, as well as in the canal in the mastoid. In the closed part of the mastoid fissure it represents the vestige of the open canal. Thus the appearance and further development of a mucosa was in strict correlation with the formation of open spaces.

The tubal orifice was wider than normal; this is unusual in cases of atresia, in which it is frequently very narrow.

Normal ossicles were reported in only 2 of the 54 cases tabulated by Marx. Only one of the observations is suggestive of the present case: Ranke found instead of the malleus and incus a single bone of cylindric shape, the stapes also being present. Tomka [6] emphasized that malformations are found most frequently in the stapes. Several authors described blending of the rudiments of the malleus and incus or formation of an ossicle suggestive of a columella. In my case the vertical position of the bone did not justify the association with a columella-like formation. The blending of malleus and incus into one ossicle is an arrest in development pointing at the latest to the sixth embryonal week; up to this stage there is no separation between the head of the malleus and the body of the incus.

There is another explanation for the presence and character of this bone. The rudimentary bone found its way into the stylomastoid foramen. The tendency of the stylomastoid process in anomalies to protrude up to a certain level into the free tympanic cavity makes it possible that the bone represents only a maximally elongated stylomastoid process. Supporting this contention is the circumstance that the bone is a simple rod without differentiation and further that it is in intimate connection with the base of the occluding plate and the structures surrounding the stylomastoid canal. Against this contention is the fact that no similar case has been reported, even with findings of every description, from the normal styloid up to building of the complete occlusion plate or penetration as a free process up to a certain level in the cavum tympani. It is impossible to give a definite analysis of this peculiar formation.

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5. Cantele, P. G.: Studio anatomo-microscopico di malformazioni dell'orecchio in un caso di idrocefalo congenito con cheilo-uranoschisi laterale, Atti d. Soc. med.-chir. di Padova **11**:949-958 (Nov.) 1933.

6. Tomka, S.: Ueber Entwicklungsanomalien des Steigbügels, Arch. f. Ohrenh. **38**:253-258 (April) 1895.

It may be a homologue of the ossicles or a prolonged styloid. It may "represent an effort of nature at compensation for the defective condition in the region."[2] In not 1 of the tabulated cases of Marx were there corresponding observations regarding the muscles of the tympanic cavity, with a degenerated tensor muscle and the stapedius without a tendon. The latter muscle showed a normal histologic picture without atrophy, which is remarkable in the absence of every possibility of function.

Regarding the early developmental date of the anomalies in the middle ear, one may disregard the possibility of fetal diseases resulting in the abnormalities found in the inner ear. The difficulties of the analysis in this particular region arise from two sources: (1) It is not always easy to differentiate arrest of development from later degeneration in the end organs; (2) the capsule of the cochlea gains its definitive form in postnatal life. One has the help of the coexistent malformations of the external and the middle ear to put the impedance for the development of at least the membranous labyrinth into an early period. The capsule of the cochlea itself, developing independently, continued on its way until normal conditions were reached. The mastoid process should be mentioned, showing the same independence of development from the other parts. It too reaches its final shape in postnatal life. In my case, however, there was a well developed mastoid, without pneumatization. Pneumatization, being a centrifugal process originating from the tympanic cavity, could not be accomplished. The complete development of the mastoid and of the osseus labyrinthine capsule indicates that the causes of arrest of development of other parts in the earliest weeks of embryonal life ceased to be effective in later periods.

The surface of the pyramid measured across the tegmen tympani was shorter than on the normal side, which is one more reason to place the date of the formative difficulties in the early embryonic period, when the pyramid begins to take shape.

The cartilaginous preformed capsule of the cochlea undergoes a development completely independent from that of the modiolus, the latter originating from the connective tissue in the axis of the cochlea, in close connection with the neural structures. The axial part of the osseous portion of the cochlea could share in this way with the soft parts in the dysgenetic process without hindering the capsule in reaching its definite normal shape and structure. The often discussed relation between a dysgenic process in the end organ and the osseous parts in immediate connection with them is an interesting counterpart to the germinal defects associated with the trophic nervous system, as put forward by Hrdlička in anomalies of the external ear. The second case of Polvogt and Crowe[6a] similarly showed absence of the modiolus above

6a. Polvogt, L. M., and Crowe, S. J.: Anomalies of the Cochlea in Patients with Normal Hearing, Ann. Otol., Rhin. & Laryng. **46**:579-591 (Sept.) 1937.

the middle turn, but without the deformed continuation of the basilar lamina in the apical turn.

The structure of the internal auditory meatus was simple, without the funnel-shaped antechamber. I have discussed the importance of the latter in certain pathologic processes.[7]

Corti's organ, together with its nerves and ganglions, is hypoplastic, arrested at an embryonal stage, as found in the second and third months. In the vestibular part a common pocket-shaped primordium of the sagittal and frontal canals is found frequently with atresia auris congenita, but here they are well developed.

Nests of spongy bone as seen in the neighborhood of the stapes in my case were repeatedly found by others together with abnormalities of the labyrinthine capsule, indicating arrested development. Manasse[8] observed cartilaginous islands near the anterior periphery of the annular ligament. They are remnants of embryologic conditions, and a connection is presumed between these islands and the later formation of otosclerotic foci. Nager[9] also found otosclerotic foci more frequent in persons with deformities in the labyrinthine capsule. Altmann and Hagen[9a] summarized the discussion concerning the development of otosclerotic tissue from such foci. In my case with fixation of the footplate any mechanical influence is excluded in promoting the formation of this condition. Altmann[10] reported the postmortem finding of a cartilaginous island in the upper wall of the internal acoustic meatus in a child aged 2 days with microtia and congenital atresia of the external auditory meatus on the left side.

The structure of the focus found near the lateral margin of the footplate demonstrates the process in a very early stage. The area is well defined, bulging at one side slightly over the wall of the labyrinthine capsule. The initial change appears in the form of very distinct blue mantles around the vessels, with compression of the latter. The resorption of the original lamellar bone progressed to complete reticulization. In the meshes of the weblike structure cartilaginous islands are enclosed

7. Kelemen, G.: Topographical Contributions to the Pathology of Neuritis Acustica, Acta oto-laryng. **21**:134-148 (Nov.) 1934.

8. Manasse, P.: Ueber Ossifikationsanomalien im menschlichen Felsenbein und ihre Beziehungen zur sogenannten Osteosklerose, Arch. f. Ohrenh. **95**:145-161 (May) 1914.

9. Nager, cited by Engström, H.: On Frequency of Otosclerosis, Acta oto-laryng. **27**:608-614 (Nov.-Dec.) 1939.

9a. Altmann, F., and Hagen, C. E., Jr.: Congenital Malformations of the Ear, in Fowler, E. P., Jr.: Medicine of the Ear, New York, Thomas Nelson & Sons, 1939, pp. 93-123.

10. Altmann, F.: Zur Kenntnis der sogenannten persistierender Knorpelherde im Bereiche der knöchernen Labyrinthkapsel, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:297 (Nov.) 1932.

with homogeneous ground substance and large, clear cells in the characteristic form of cartilage cells. As there is no known relation between the development of otosclerotic foci and a pathologic change in any other part of the hearing organ, one cannot bring this focus into organic relationship with the anomalies in its vicinity. The age of 10 years would be the proper time to look for the beginning of the process in the labyrinthine capsule. It must be emphasized that the fixation of the plate of the stapes is unrelated to this focus. The ankylosis represents an arrest of development, as the plate of the stapes differentiates from the capsule of the labyrinth, in contrast to the crura. The latter originate from the second branchiogenic arch; their faulty attachment to the footplate, as demonstrated, illustrates the different origin.

All the essentials of the roentgen report were confirmed by the microscopic observations.

The question of the possibility of hearing in early life which may have been lost later by progressive degeneration should be answered in a negative way. The absence of the cochlear duct makes hearing unlikely. Whereas the beginning of pneumatization from the tubal ostium may have progressed some time later, an air-conducting apparatus could never form. The fact that both windows were closed, the oval window by bony fixation of the plate and the round window by a solid fibrous wall, according to present day concepts of hearing excludes the likelihood of even bone conduction.

As I have discussed the microfissures of in vivo origin elsewhere,[10a] it is sufficient to mention in connection with the present observations that Barth [11] demonstrated them in a case of malformation of the ear, in which there was histologic examination. Less pneumatization than normal explains the undamaged condition in a fractured cranial base.

It was found several times, e. g. in the operations reported by Colver,[12] that in the absence of a tympanic membrane the canal reached the bony occlusive plate forming the lateral tympanic wall. Such was the case here, but with this important difference: The canal rudiment was formed only in its medial half. The cavity reminded one in some respects of the observation in the case of Novick,[13] in which there was a successful operation. With resorption of the exostosis at a later period, as happens with these changeable structures, a considerable lumen may

10a. Kelemen, G.: Ueber die Fissuren im knöchernen Innenohr, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:36-49 (Nov.) 1933.

11. Barth, H.: Ueber eine anatomische Anomalie des Bogengangsapparates, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:375-379 (May) 1932.

12. Colver, B. N.: Bilateral Atresia of the External Auditory Canal Without Malformation of the Concha [fig. 7], Arch. Otolaryng. **17**:476-483 (April) 1933.

13. Novick, N. J.: Atresia of the External Auditory Meatus, Arch. Otolaryng. **30**:744-748 (Nov.) 1939.

have been formed. But even so the operative opening of the inner bony occlusion would be a serious obstacle to overcome in this case.

The opinion is unanimous not to try to find and reconstruct the external canal. Herczel and Krepuska [14] wrote as far back as 1895, in their comment on a case of successful creation of a new canal across the mastoid, that it is futile to look for something in a place where it does not exist. This seems to be the general opinion almost forty years later, as confirmed by the statement of Richards. In the present case the operation with an attempt to form a canal through the mastoid would have uncovered a preformed canal at this unusual location. The artificial widening of it could have been done without difficulty. Wanamaker [15] operated successfully, creating an artificial opening of the cortex and finding a preformed canal but no external auditory meatus or drum membrane; but as there was suppuration the comparison cannot be assumed. The possibility of maintaining an open passage is questionable in this unique case of a canal crossing a mastoid untouched by any inflammatory process.

SUMMARY

A description is given, with microscopic examination of serial sections, of a malformation in the external, the middle and the internal ear on the right side in a 10 year old girl. The case shows some hitherto unreported features. The tympanic membrane, the vestibular membrane (Reissner), the tectorial membrane, the tympanic branch of the glossopharyngeal nerve (Jacobson), the tendon of the stapedius muscle and the chorda tympani were completely absent. The auricle, the external auditory canal, the malleus and the incus (both replaced by a single bone), the stapes, the cochlear aqueduct, the tensor tympani muscle, the modiolus cochleae and all the neural end organs of the inner ear were represented only by rudimentary structures.

The deformities were partly arrests of development and partly true malformations. They were completed possibly in the first two months of embryonal life. The etiologic factor responsible for the agenesis ceased to exist after this period. Thus the mastoid process and the labyrinthine capsule, formed later, were able to reach a high developmental stage, although the former was without pneumatization and the latter was somewhat reduced in size.

Near the lateral circumference of the annular ligament an otosclerotic focus originated in the area of an embryonal cartilaginous island.

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14. Herczel, M., and Krepuska, G.: Atresia congenita meatus auditorii externi, Gyógyászat **35**:241-243 (May) 1895.

15. Wanamaker, A. T.: Mastoiditis in Congenitally Deformed Ears, Ann. Otol., Rhin. & Laryng. **48**:140-147 (March) 1939.

STAPHYLOCOCCIC MENINGITIS FROM HIPPOCRATES TO LEGENDRE AND BEAUSSENAT

A MORTAL DISEASE

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Staphylococcic meningitis is a disease of such malignancy that every carefully observed case terminating in recovery should be fully reported to aid in the care of future victims of this malady. We intend to present the records of a considerable number of patients who have suffered from this disease, including several who survived. These are patients who have received our bacteriophage service during the last ten years. Before proceeding to the discussion of our own experience in this field we purpose to undertake a review of the more significant recorded observations from the time when drying of the spinal marrow and accumulation of bile were regarded as the causes of spasms and paralysis, through the period when purulent exudate in the membranous coverings of the brain and cord was recognized as a feature of these disorders and the later period, when the infectious activity of specific microbes came to be appreciated in the causation, to the modern era, in which precise recognition of the etiologic agent, be it spirochete, mycobacterium, neisseria, streptococcus, staphylococcus or filtrable virus, supplies the most significant evidence on which to base prognosis and therapy.

Throughout the review an attempt will be made to focus the attention on staphylococcic meningitis, more or less definitely recognizable as such, and in the later years to give attention particularly to the examples of recovery.

The signs and symptoms of meningitis can be recognized only with difficulty in the descriptive writings of Hippocrates, where passages which may relate to meningitis are obscured by association with descriptions of signs which are obviously unrelated, such as the following:

The third form of consumption. In this form the patient's spinal marrow becomes full of blood and bile. Likewise he wastes away also from the hollow veins,

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which become filled with watery slime and with bile. The manifestations are the same when the consumption is due to either of these two alterations. The spinal marrow dries up especially when the veins leading into the marrow and the entrance from the brain become obstructed. Resultant injury causes the patient to become ill and to suffer the sequelae. The spinal marrow dries out. Venereal reaction ceases. The patient suffers the following: severe pain afflicts the head, the neck, the lumbar region, the lumbar muscles and the joints of the thighs, so that often they cannot be bent; also the excrement does not pass out but remains stagnant, and also the passage of urine is difficult. . . . The disease is severe (with high mortality).[1]

Possibly this passage may relate to tuberculous meningitis. In another section Hippocrates seems to have included along with genuine tetanus some forms of meningitis.

Opisthotonus. In regard to opisthotonus the patient in addition to the usual manifestations (of tetanus) generally shows further signs. This arises when the disease attacks the posterior tendons of the neck. It may sometimes come from angina and sometimes from swelling of the uvula and sometimes from suppuration of the jaws on both sides; at times also it comes from the cranial cavity, if after fever has set in, the spasm appears; this disease has also appeared as a result of wounds. The victim is drawn backwards, the back and the chest are painful and the patient groans. The spasms are so powerful that the attendants are hardly able to hold him to prevent falling out of bed.

Is this not meningitis due to extension of facial and pharyngeal suppuration, or meningitis of cryptogenic origin, that is, from the cranial cavity?

Another tetanus. This kind of tetanus is less dangerous than the preceding. It arises from various causes, but many patients have acquired the disease following a blow on the back. The entire body may be seized with cramps, or sometimes only one part, wherever it may be. At first the patient may walk about but later he is confined to bed, when the pain and spasm diminish. If he gets up again he may get about for a few days but soon takes to his bed again because of the same pain. For a long time there may be remissions and exacerbations.[2]

This passage suggests the lighting up of a latent vertebral osteomyelitis by trauma, with later extension to the spinal meninges.

The obscurity in respect to diseases of the cranial contents and of the spinal marrow persisted until the era of pathologic anatomy, when important observations were made in France, in the British Isles, in Austria, in Germany and at Padua in Italy. Dease[3] in 1776 advocated

1. Kapferer, R.; Sticker, G., and others: Die inneren Krankheiten, in Die Werke des Hippokrates: Die hippokratische Schriftensammlung in neuer deutschen Übersetzung, Stuttgart, Hippokrates-Verlag, 1936, pt. 19, chap. 13, pp. 42-43. The text above is a free translation from the German.

2. Kapferer, Sticker and others,[1] chaps. 52, 53 and 54, pp. 93-95. The text above is a free translation from the German.

3. Dease, W.: Observations on Wounds of the Head with a Particular Enquiry into the Parts Principally Affected in Those Who Die of Such Injuries, London, G. Robinson, 1776.

the delayed suture of contaminated lacerations of the scalp in order to avoid destructive suppuration and sloughing. His first case report is that of a boy of 17 who was wounded by a blow on the frontal region on Aug. 24, 1766. After two weeks (September 7) he was seized with smart shivering and persistent nausea, headache and fever, and he raved occasionally. A large trephine opening on September 8 released a collection of pus situated between the skull and the dura. The dura was locally detached and sloughy. The patient recovered and was discharged November 2. Evidently the dural inflammation was still localized at the time of the operation. In most of his patients, however, Dease found that the symptoms of meningitis becoming evident many days after cranial injury were often related to a deeper inflammation spreading widely in the arachnoid, pia and superficial brain substance and sometimes associated with protrusion of one eye and puffing of the conjunctiva to a fungus-like prominence. In these instances operation was not successful and may even have contributed to the unfortunate outcome. Doubtless staphylococcic infection played an important role in some of the 25 patients whose cases were reported by Dease.

Frank [4] expressed dissatisfaction with the confusion which recent anatomic studies had revealed in the ideas and terminology based on clinical observations of diseases of the brain and spinal cord. At Pavia progress was being made by (1) dissection of the human body in its natural state, (2) dissection of all sorts of animals and (3) dissection of the human body in its manifold unnatural (pathologic) states.

Baillie [5] in 1793 recognized among diseases of the dura: (*a*) congestion, (*b*) presence of coagulable lymph, (*c*) suppuration, (*d*) erosion, (*e*) scrofulous tumors, (*f*) spongy tumor, (*g*) ossification and (*h*) strong cranial adhesions. Among alterations of the arachnoid he listed: (*a*) congestion, (*b*) suppuration, (*c*) adhesions and (*d*) scrofulous tumors and hydatids; and in the brain, abscesses.

The results of many observers were summarized by Vogel [6] in a most satisfactory fashion. He stated that inflammation may be located either in the brain membranes or on the surface of the cerebellum or cerebrum or, less often, deeper in the brain substance and that it may extend into the spinal marrow. The descriptions of signs and symptoms are excellent even for the present time. He suggested trephining when pus

4. Frank, J. P.: Untersuchungen über die Krankheiten des Rückrats und des in ihm befindlichen Rückmarks, Sammlung auserlesener Abhandlungen zum Gebrauche praktischer Aerzte, Leipzig, Dyckischen Buchhandlung, 1792, vol. 15, pt. 1, pp. 260-306.

5. Baillie, M.: The Morbid Anatomy of Some of the Most Important Parts of the Human Body, London, J. Johnson, 1793.

6. Vogel, S. G.: Handbuch der practischen Arzneywissenschaft, Stendal, D. C. Franzen u. Grosse, 1795, pt. 4.

is present and not naturally evacuated. These publications in the last decade of the eighteenth century recorded a clear recognition of inflammatory disease of the meningeal membranes as distinct from disease of the brain and provided a foundation for attempted differential diagnosis between tetanus, tuberculous disease and syphilitic disease on the one hand and seropurulent and purulent meningitis on the other. Brera,[7] who was associated with Frank at Pavia, recognized inflammation of the spinal meninges as distinct from disease of the spinal cord. Another author of the school at Padua, Bergamaschi,[8] made this differential diagnosis even more distinctly. His sixth case, that of a boy of 13 with caries of the twelfth thoracic vertebra and meningeal inflammation extending from this vertebra down to the last lumbar, would appear to have had fulminant staphylococcic meningitis complicating vertebral osteomyelitis.

Abercrombie[9] in 1818 clearly described cerebrospinal meningitis following chronic otitis media. He also described a patient of Mr. Charles Bell who had purulent spinal meningitis evidently taking origin from caries in the cartilage between the seventh cervical and the first thoracic vertebra, following trauma. The description suggests that this was an example of staphylococcic meningitis. The same author[10] has described several examples of cerebral meningitis, associated with abscess of the brain in some instances. His descriptions of cases indicate a clear recognition of derivation from inflammations of the ear, mastoid and ethmoid and from cranial osteomyelitis and puncture of a fontanel.

Parent-Duchatelet[11] and Martinet in 1821 tabulated 116 cases of arachnoiditis in their preliminary group and considered additional examples of this condition. Some of these were undoubtedly tuberculous; others were examples of epidemic meningitis and still others of genuine tetanus, as for example observation 119. The descriptions are so adequate that staphylococcic meningitis can be diagnosed with some confidence in observations 18 and 19, both of which are cases of purulent cerebral leptomeningitis subsequent to wounds of the scalp complicating cranial fracture.

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7. Brera, V. L.: Della rachialgite, Leghorn, Presso Tommaso Masi & Co., 1810.

8. Bergamaschi, G.: Osservazioni sulla infiammazione dello spinale midollo e delle sue membrane, Pavia, Bolzani, 1810.

9. Abercrombie, J.: Observations on Diseases of the Spinal Marrow, Edinburgh M. & S. J. **14**:42-68 (Jan. 1) 1818.

10. Abercrombie, J.: Observations on Chronic Inflammation of the Brain and Its Membranes, Edinburgh M. & S. J. **14**:265-328 (July 1) 1818.

11. Parent-Duchatelet, A. J. B., and Martinet, L.: Recherches sur l'inflammation de l'arachnoïde cérébrale et spinale, Paris, Crevot, 1821.

In the monograph of Ollivier[12] one can recognize a description which might very well fit staphylococcic meningitis. A man of 35 (observation 70) had an abscess of the face about a diseased tooth in June 1826. After seventeen days his neck became immovable and severe pain developed in the lumbar and abdominal regions and in the legs. He died suddenly on the twentieth day. Autopsy disclosed flaky fluid in the fourth ventricle with a yellowish pseudomembrane over its roof. There was creamy pus in the lumbar muscles, which could be traced to its exit between the laminas of the third and fourth lumbar vertebrae. Within the spinal canal the purulent exudate extended between the arachnoid and the pia and was most abundant from the ninth thoracic vertebra to the lower lumbar region.

Abercrombie[13] in 1828 clearly distinguished inflammations of the cranial dura: (1) idiopathic, (2) connected with disease of the ear and caries of the temporal bone, (3) related to disease of the nose, (4) related to caries of the cranium and (5) associated with obliteration of the lateral sinus. He also recognized inflammations of the arachnoid and of the pia mater in various locations, and spinal meningitis. As an instance of meningitis of the cord he reported the case of a man aged 26 (patient of Duncan) with chronic aural disease who began to have headache early in April and died on April 18, 1817. There was pus all along the cord between it and its membranes. Another patient of Duncan had greenish yellow coagulable lymph on the posterior surface of the cord and on the surface of the brain.

Without reference to the earlier literature, Albers[14] described inflammation of the spinal dura. The anatomic observations in his cases do not suggest staphylococcic infection.

Constant[15] in 1835 gave a detailed clinical and anatomic description of the case of Génévière Chapagniol, aged 10 years, which is highly suggestive of a staphylococcic spinal meningitis involving the dura and arachnoid and extending to the cerebellar region. The purulent exudate extended into the sheaths of many spinal nerves.

In the magnificent atlas of Cruveilhier[16] this author recorded the case of Mlle. Gault, aged 18, who suffered a lameness in the left leg

12. Ollivier, C. P.: Traité de la moelle épinière et de ses maladies, ed. 2, Paris, Chevot, 1827, vol. 2, pp. 569-575.

13. Abercrombie, J.: Pathological and Practical Researches on Diseases of the Brain and Spinal Cord, Edinburgh, Waugh & Innes, 1828.

14. Albers: Die Entzündung der harten Haut des Rückenmarks, Perimeningitis medullae spinalis, J. d. Chir. u. Angenh. **19**:347-362, 1833.

15. Constant, T.: Hôpital des Enfans Malades. Revue de la clinique de M. Baudelocque, médecin de cet hôpital pendant les mois d'Octobre, Novembre et Décembre 1834, Gaz. méd. de Paris **3**:70-74 (Jan. 24) 1835.

16. Cruveilhier, J.: Maladies de la moelle épinière, in Anatomie pathologique du corps humain, Paris, J. B. Baillière, 1835-1842, vol. 2, pt. 32, pp. 7-12.

followed by paralysis and pain. She was treated with moxas and by bloodletting and recovered after a long illness with hardly evident slight disability. The author regarded this as a recovery from spinal meningitis. Of course there was no bacteriologic study. The clinical description suggests poliomyelitis.

Tatum [17] reported the postmortem observation of purulent spinal meningitis extending up to the middorsal region subsequent to puncture of a congenital lumbosacral meningocele. The description suggests infection by staphylococci or colon bacilli and illustrates the confusion of meningitis with tetanus which still persisted in 1852.

Gangrenous extension of sacral decubitus to perforate the spinal dura and give rise to seropurulent meningitis of the cord and the base of the brain was reported by Duchek.[18] This may have been a staphylococcic meningitis.

Wood [19] reported an example of meningitis and intracranial thrombophlebitis following a slight scalp wound which had healed. Suppuration arose beneath the scar, and death took place three weeks after the injury. At autopsy there was found a fracture of the inner table of the skull, with pus between the dura and the bone, in the superior longitudinal sinus and in the pia beneath the site of the wound. There were also secondary purulent deposits in the lungs. One has very little reserve in accepting this as an example of staphylococcic infection.

A man aged 30 was reported on by Bristowe.[20] At autopsy the spinal theca was found to be enveloped in pus, which extended also into the arachnoid sac, upward to the cranial cavity and laterally around the spinal nerve roots to produce a series of abscesses along each side of the spine. Again one recognizes the probability of staphylococcic meningitis.

Gull [21] reported the case of a man of 23 in whom spinal meningitis developed after exposure to cold and wet and who died on the thirteenth day (case 4). Epidural purulent exudate extended the entire length of the spine, and there was pus around the spinal nerves at their exits.

The book of Reeves [22] did not record any example of apparent staphylococcic meningitis, but it did suggest rather definitely the

17. Tatum: Supposed Spina Bifida in a Young Man: Enormous Size of the Tumour; Puncture; Tetanus; Death; Autopsy, Lancet **2**:56-57 (July 17) 1852.

18. Duchek, A.: Bericht über Prof. Hamernik's Abteilung des allg. Krankenhauses zu Prag während der Zeit vom 1 October 1850 bis letzten September 1851, Vtljschr. f. prakt. Heilk. **10**:1-97, 1853.

19. Wood: Slight Injury to the Head, Followed by Meningitis, M. Times & Gaz. **8**:473-474 (May 6) 1854.

20. Bristowe: Suppurative Inflammation of the Spinal Theca, M. Times & Gaz. **10**:19 (Jan. 6) 1855.

21. Gull, W.: Cases of Paraplegia, Guy's Hosp. Rep. **2**:143-187, 1856.

22. Reeves, C. E.: Diseases of the Spinal Cord and Its Membranes, London, Simpkin, Marshall & Co., 1858.

existence of a peculiar form of meningeal irritation deserving the designation of epidemic meningitis, thus foreshadowing the bacteriologic era.

Traube [23] in 1863 reported two examples of purulent spinal pachymeningitis, with evidence of erosion of vertebral transverse processes in 1 patient. The possibility of a primary osteomyelitis was mentioned in the discussion.

Müller [24] reported in 1868 the case of a man 26 years old who at autopsy had purulent exudate between the dura and the bone of the vertebral column and also extensions of the exudate along the spinal nerves. He named the condition peripachymeningitis.

A well known example of purulent spinal peripachymeningitis, apparently of staphylococcic causation, was that of Lewitsky.[25] A man aged 44 suffered an injury to the lumbosacral region on January 12, but continued at work three days, then fell and was helped into the house. Paresis was succeeded by total paraplegia and loss of sphincter control, with eventual death on February 6. The spinal dura was surrounded by pus with extension to the subdural space and to a lesser extent to the cranial meninges.

Lemoine and Lannois [26] recorded a somewhat similar observation in 1882. A soldier, aged 22, fell on his hands. Three days later there was ascending paraplegia followed by death in seven days. There was a thick layer of pus outside the spinal dura but none within the dural sheath.

Benson [27] in 1884 reported meningitis following enucleation of a shrunken eyeball. There was extension of purulent exudate along the optic nerve.

Epidemic meningitis had meanwhile forced itself on the attention of physicians because of local outbreaks in various parts of Europe.

The pioneer bacteriologic report by Leichtenstern [28] in 1885 recorded the observation of cocci, single and grouped, resembling gonococci in arrangement, mostly within leukocytes but occasionally outside. The

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23. Traube, L.: Meningitis spinalis, Deutsche Klin. **15**:198-200 (May 16) 1863.

24. Müller, R. H.: Peripachymeningitis, Thesis, Königsberg, Gruber u. Longrien, 1868.

25. Lewitsky, P.: Ein Fall von Peripachymeningitis spinalis, Berl. klin. Wchnschr. **14**:227-230 (April 23) 1877.

26. Lemoine, G., and Lannois, M.: Périméningite spinale aiguë, Rev. de méd. **2**:533-544, 1882.

27. Benson, A.: Meningitis After Enucleation of Eyeball, Lancet **1**:803 (May 3) 1884.

28. Leichtenstern, O.: Ueber Meningitis cerebro-spinalis epidemica, Deutsche med. Wchnschr. **11**:391-392 (June 4); 537-540 (July 30) 1885.

results from his cultures were not convincing and his experiments on animals evidently failed. However he suggested the name "meningococcus."

Netter [29] in 1887, following the observations of Klebs, of Eberth, of Fränkel and of Foà and Bordoni-Ufreduzzi, all of whom suggested the causation of meningitis by the pneumococcus, was able to demonstrate conclusively the importance of the pneumococcus of Fränkel in the causation of purulent meningitis. Unfortunately he also ascribed to this organism the essential etiologic role in epidemic cerebrospinal meningitis.

Later in the same year Weichselbaum [30] confirmed the observations of Leichtenstern and was successful in obtaining satisfactory cultures of the meningococcus and in causing fatal infection in mice. Subsequent investigations have abundantly proved the causal relation of the meningococcus in epidemic meningitis.

Galippe [31] appears to have been the first to report the actual demonstration of Staphylococcus aureus as the causative agent of meningitis. A severe suppuration of the left side of the lower jaw with multiple fistulous tracts developed in a man aged 23 who had a large impacted wisdom tooth. Cultures of the exudate yielded growth of Staph. aureus, a finding which was confirmed by Netter. Intravenous inoculation of a rabbit caused death with multiple purulent infarcts in the kidneys and some pulmonary lesions. Meningitis suddenly developed, and the patient died quickly. There was no autopsy. However, the physicians, Galippe and Simon, agreed that the meningitis was caused by the staphylococcus.

In the following year Netter [32] himself observed a mixed infection, with pneumococci and staphylococci in the meningeal exudate, following a suicidal bullet wound through the mouth. This was doubtless the first actual demonstration of the staphylococcus in meningeal exudate within the cranium.

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29. Netter: De la méningite due au pneumocoque (avec ou sans pneumonie), Arch. gén. de méd. **19**:257-277 (March); 434-455 (April) 1887; **20**:29-51 (July) 1887.

30. Weichselbaum, A.: Ueber die Aetiologie der akuten Meningitis cerebrospinalis, Fortschr. d. Med. **5**:573-583 (Sept. 15); 620-626 (Oct. 1) 1887.

31. Galippe, V.: Du rôle des parasites infectieux dans la génèse des accidents de l'évolution de la dent de sagesse, J. d. conn. méd. prat. **9**:314-316 and 321-324. 1889.

32. Netter: Méningite suppurée consécutive à un coup de révolver; coexistence du pneumocoque et du staphylococcus pyogènes aureus; pénétration directe dans la cavité crânienne de microbes pathogènes préexistant dans la bouche, Compt. rend. Soc. de biol. **2**:140-142 (March 8) 1890; J. d. conn. méd. prat. **10**:125-126 (April 17) 1890.

Quincke [33] in 1891 introduced the important procedure of spinal puncture, primarily for therapy. This has become of inestimable value in the diagnosis of meningitis and in the recognition of the infectious agent concerned.

Antony [34] in 1892 reported an example of cryptogenic staphylococcic spinal meningitis, acceptable without reserve. A robust soldier, aged 23, with an excellent record, became constipated. On Oct. 12, 1889 cramps developed in his legs, followed by loss of motility and sensation below the umbilicus. On October 16 the paralysis had extended to the upper extremities and sévere pain in the neck and head appeared, with occasional delirium, followed by death at 6 a. m. October 18. The cranial meninges were intensely congested but without exudate. The spinal dura from the third cervical vertebra to the lumbar level was covered with thick gray pus located between the dura and the vertebral arcs. Pus was not found within the dural sac, but the vessels here were engorged and the cord was softened. The pus yielded Staph. aureus in pure culture. The cerebral fluid was sterile. Antony suggested that trephining the spine to relieve pressure should be done in such a case and that the operation might succeed. Netter, in the discussion of Antony's presentation, suggested that the disease had originated in a latent focus of staphylococcic infection, just as active osteomyelitis may appear after a long latent period.

Le Gendre and Beaussenat [35] in the same year reported on a man aged 47 who was suddenly seized with violent hemicrania and pain in the right ear. On the following day there was an abundant flow of pus from the ear and less pain. The headache persisted, with quiet delirium and a temperature of 40.4 C. (104.7 F.). On the sixth day the right knee became painful and swollen, and on the eighth day pus was aspirated from it. Death occurred on the ninth day, with the patient in coma following violent delirium. The autopsy revealed suppurative basilar meningitis and purulent arthritis. Staph. aureus was found in pure culture in the ear, meninges and knee joint.

COMMENT

We have attempted in this paper to trace by brief mention of the most important observations the knowledge of staphylococcic meningitis

33. Quincke, H.: Ueber Hydrocephalus, Berl. klin. Wchnschr. **28**:549 (June 1) 1891; Die Lumbalpunction des Hydrocephalus, ibid. **28**:929-933 (Sept. 21); 965-968 (Sept. 28) 1891.

34. Antony: Périméningite à staphylocoques dorés et pseudo-rheumatisme infectieux à streptocoques pyogènes, Bull. et mém. Soc. méd. d. hôp. de Paris **9**:74-82, 1892.

35. Le Gendre and Beaussenat: Infection staphylococcique: Otite, méningite et arthrite suppurées, broncho-pneumonie; mort, Bull. et mém. Soc. méd. d. hôp. de Paris **9**:577-580 (July 22) 1892.

from the obscurity of the early clinical observations of Hippocrates [1] through the stage of precise anatomic localization of the morbid process in the coverings of the brain and cord by Dease,[3] Baillie,[5] Frank,[4] Vogel [6] and others in the eighteenth century to the later demonstration of the specific bacterial causes of some of the various types of meningitis in the early days of the bacteriologic era and the eventual reliable recognition of staphylococci as the infectious agents in certain examples of cranial and spinal meningitis by Galippe,[31] Netter,[29] Antony [34] and Le Gendre and Beaussenat.[35]

The careful clinical observations of the older writers, viewed in the light of modern knowledge, permit recognition, with reasonable probability, of staphylococcic meningitis as a sequel to suppuration of the jaws and as a complication of vertebral caries in the time of Hippocrates. Doubtless very few will even question the malignant activity of the staphylococcus in the fatal infections following cranial wounds recorded by Dease [3] (1776). Brera's [7] (1810) case 6 could hardly be other than an instance of staphylococcic spinal meningitis and the same diagnosis seems warranted for the patient of Mr. Charles Bell, reported on by Abercrombie [9] (1818). Eventually the bacteriologic studies of Netter and his associates served to identify staphylococcic meningitis with certainty.

Previous to 1893, there were only feeble suggestions in regard to possible recovery from meningitis of this type. Dease [3] (1776) had observed recovery following trephine opening of a localized epidural abscess but could record no success in the more diffuse inflammations of the pia-arachnoid. Cruveilhier [16] (1835-1842) recorded a recovery from supposed spinal meningitis, for which the diagnosis of meningitis cannot be accepted. As we shall see in the subsequent paper, the claimed recoveries from staphylococcic meningitis became more evident and more credible in the last decade of the nineteenth century, with gradual improvement in the prognosis during the subsequent half-century and a still less gloomy outlook for the future.

EFFECT OF THE NEWER SULFANILAMIDE DERIVATIVES ON THE NASAL MUCOSA OF RABBITS

PRELIMINARY REPORT

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LOS ANGELES

In the last five years sulfanilamide and its derivatives have revolutionized the treatment of many serious bacterial infections. The spectacular results obtained when these drugs were administered internally prompted many clinicians to apply them locally. This allows a greater concentration at the site of infection. Favorable results reported in the healing of infected wounds,[1] compound fractures [2] and many other conditions [3] led my associates and me to become interested in investigating the value of the drugs for the local treatment of infections of the nose and paranasal sinuses.

For the past eighteen months we have used a saturated (0.8 per cent) solution of sulfanilamide [4] for the treatment of sinusitis, the solution being introduced into the infected sinuses by the Proetz displacement method. Some apparent success was noted, but it was thought that a stronger solution might be more efficacious, were it available.

Recently solutions of greater strength have been made possible by the introduction of the sodium salts of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) and sulfadiazine (2-[paraaminobenzene-

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From the Department of Otolaryngology of the Santa Rita Clinic.

Read at the regular meeting of the Los Angeles County Medical Association, Jan. 15, 1942.

1. Loeb, W. A., and Thornton, J. S.: Neoprontosil and Sulfanilamide in the Local Treatment of Infected Wounds, M. Bull. Vet. Admin. **18**:65-71 (July) 1941. Donahue, C. D.: The Use of Sulfanilamide in Infected Genito-Urinary Wounds, J. Urol. **46**:562-566 (Sept.) 1941. Mayo, C. W., and Miller, J. M.: Solution of Sulfanilamide in the Local Treatment of Infected Wounds, Proc. Staff Meet., Mayo Clin. **15**:609-611 (Sept. 25) 1940.

2. Jensen, N. K.; Johnsrud, L. W., and Nelson, M. C.: The Local Implantation of Sulfanilamide in Compound Fractures, Surgery **6**:1-12 (July) 1939.

3. Crile, G., Jr.: The Local Use of the Sulfonamide Drugs, Cleveland Clin. Quart. **8**:149-153 (July) 1941. Bickford, B. J.: Local Sulphanilamide for Penetrating Wounds of the Joints, Brit. M. J. **1**:627 (April 26) 1941. Key, J. A., and Frankel, C. J.: The Local Use of Sulfamethylthiazole, Ann. Surg. **113**:284-297 (Feb.) 1941. Lockwood, J. S.: Sulfonamide Therapy as an Aid to Surgery, Surg., Gynec. & Obst. **72**:307-311 (Feb., no. 2A) 1941.

4. The solution was supplied by Don Baxter, Inc., Glendale, Calif.

sulfonamido]-pyrimidine), which are readily soluble in distilled water. However, these drugs are highly alkaline. An isotonic solution (4.7 per cent) of each was prepared and its p_H determined by use of an electric p_H meter. The p_H of the solution of sodium sulfathiazole was 9.67,[5] and the p_H of the solution of sodium sulfadiazine was 9.87.[5]

Because of the highly alkaline nature of these solutions, we decided to investigate also the possibilities of butanoylsulfanilamide, a newer member of the sulfanilamide group, which has not yet been released for clinical use. An isotonic solution of this chemical prepared with distilled water and sodium hydroxide was found to have a p_H of 7.3,[5] just slightly above the neutral point.

The soluble nature of these newer derivatives of sulfanilamide seemed to solve the problem of providing solutions of sufficient strength to be highly efficacious for intranasal use, but on account of a possible toxicity or a caustic action resulting from the strongly alkaline character of sodium sulfathiazole and sodium sulfadiazine, it was deemed advisable to determine the effects of these drugs on the normal mucous membranes of animals before attempting their use in human beings.

The rabbit was thought to present a mucosa more nearly comparable to that of human beings than the mucosa of any other laboratory animal. The rabbit has large nasal accessory sinuses, and of these the maxillary sinuses are a part of the nose. They are readily accessible to nose drops given in the usual manner. The rabbit also has a large area of olfactory mucous membrane in the posterior-superior portion of the nose which is probably more susceptible to injury than that of a human being. The nasal mucosa is comparable histologically to that of the human being. In the present study 21 animals were used, 3 in each experiment.

Before starting the drug experimentation, we felt it necessary to determine the amount of mucosal surface covered by the nose drops. One animal was selected, and 10 drops of a 1 per cent solution of gentian violet was placed in each nostril. At autopsy the entire mucous membrane of the nose and portions of that of the bronchial tree and lungs were found to be stained with the dye.

Further, since in most cases the use of a vasoconstrictor in conjunction with the sulfanilamide derivative would be indicated for relief of the nasal congestion and to assure the accessibility of the entire nasal cavity to the solution, we decided to employ one of the commonly used shrinking agents. A solution containing neo-synephrin hydrochloride [5a]

5. The p_H was determined by Dr. A. O. Beckman, of the National Technical Laboratory.

5a. The solution used (isophrin, supplied by Broemmel Pharmaceuticals, San Francisco) is an isotonic solution of neo-synephrin hydrochloride (0.25 per cent) in Ringer's solution to which are added 0.005 per cent of a combination of menthol and eucalyptol and 1 : 64,000 parts of phenol. It has a p_H of 4.5.

was selected, as it is the preparation we have prescribed for home use and have used in the office as a nasal spray and for Proetz displacement treatment for the past three years. Clinically, it is less irritating and more pleasant than most nasal medicaments and is less toxic than ephedrine, according to Tainter and Stockton.[6] Many patients use this type of drug frequently in the early stages of acute rhinitis; so it was thought advisable to ascertain its effect if used as much as ten times a day for a short period.

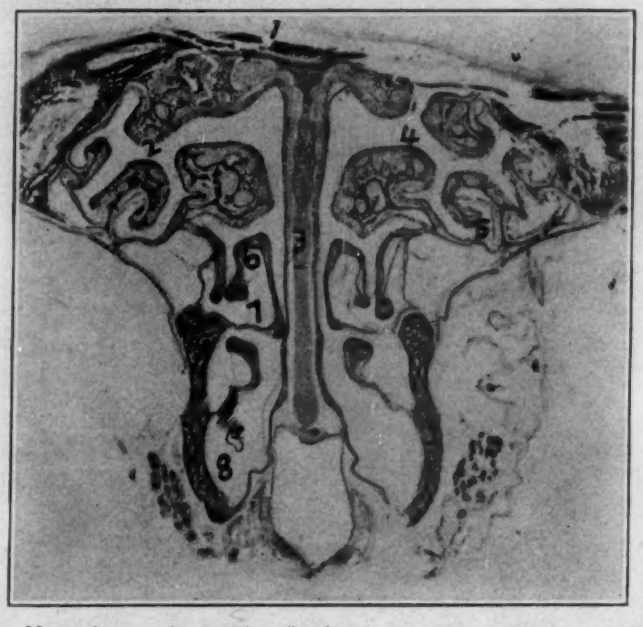

Fig. 1.—Normal nose of a rabbit. Section from posterior block. *1* indicates the nasal bone; *2*, the ethmoid cells; *3*, the nasal septum; *4*, the superior turbinate (the nasoturbinal); *5*, the middle turbinate (the ethmoturbinal proper); *6*, the inferior turbinate (the maxilloturbinal); *7*, the vomeronasal organ, and *8*, the maxillary sinus.

DESCRIPTION OF EXPERIMENTS

Ten drops of one of the following solutions was instilled in each nostril as follows:

Sodium sulfathiazole (4.7 per cent) three times daily for ten days (series 1).

Sodium sulfathiazole (4.7 per cent) three times daily for thirty days (series 2).

6. Tainter, M. L., and Stockton, A. B.: Comparative Actions of Sympathomimetic Compounds: The Circulatory and Local Actions of the Optical Isomers of Meta-Synephrin and Possible Therapeutic Applications, Am. J. M. Sc. **185**:832-843 (June) 1933.

Sodium sulfadiazine [7] (4.7 per cent) three times daily for thirty days (series 3).

Butanoylsulfanilamide [7] (4.7 per cent) three times daily for thirty days (series 4).

Solution containing neo-synephrin hydrochloride [5a] three times daily for ten days (series 5).

Fig. 2.—Normal nasal mucosa of a rabbit. Section of a vomeronasal organ from the anterior block.

Solution containing neo-synephrin hydrochloride [5a] ten times daily for three days (series 6).

Immediately after the last instillation, the animals were killed by intravenous injection of air. The head was fixed at once in solution of formaldehyde U. S. P. After fixation, the nose was removed in one block and embedded in paraffin.

7. This drug was supplied by Dr. M. L. Crossley, of the Calco Chemical Division of the American Cyanamid Company, Bound Brook, N. J.

Twelve blocks were made on the nose serially. Sections were made from each block in an attempt to determine the condition of the mucosa throughout the nose. The sections were studied especially for the condition of the mucosa and for evidence of injury or destruction.

Three untreated, apparently normal animals were killed and their nasal mucosa studied serially as described in the foregoing paragraph for controls. Figure 1

Fig. 3.—Normal nasal mucosa of a rabbit. Section of the tip of a middle turbinate from the posterior block.

shows the entire block section. Figure 2 is a photomicrograph of the anterior section and figure 3 of the posterior section.

RESULTS

There seemed to be considerable discomfort after prolonged intranasal application of the sulfanilamide derivatives. This was evidenced by great restlessness and audible sounds of distress after instillation.

After six days of treatment the mucocutaneous junction of the external nares in all the animals of the series treated with butanoylsulfanilamide showed redness, some excoriation and other evidences of severe irritation.

At autopsy, in all of the animals treated with sulfanilamide derivatives there was evidence of inflammation throughout the nose. This seemed

Fig. 4.—Rabbit's nose treated with sodium sulfathiazole. Section of a vomeronasal organ from the anterior block, showing round cell infiltration and desquamation of epithelium.

to be more marked in those in which sodium sulfathiazole and butanoylsulfanilamide were used. The posterior portion of the nose was found to be filled with a grayish exudate. The mucous membranes were markedly thickened.

Microscopic study of the sections from the animals in the series treated with sodium sulfathiazole and the series treated with butanoyl-

sulfanilamide showed injury to the entire nasal mucosa. In the anterior portion this consisted of degeneration of the epithelial layer in many areas. The intensity and distribution of the destruction increased as the posterior portion of the nose was approached. The destruction seemed greatest in the olfactory area. The last three sections showed complete necrosis and sloughing of all soft tissue down to the bone and

Fig. 5.—Rabbit's nose treated with sodium sulfathiazole. Anterior tip of a middle turbinate from the posterior block, showing destruction of all soft tissues.

cartilage, with no areas of normal mucosa. We were unable to find any evidence of injury to the bone or cartilage, although there were some areas where the perichondrium was swollen and desquamated. The sinuses were filled with abundant exudate, consisting of degenerated epithelial cells, round cells, a few giant cells and masses of debris. All of the sections from all of the animals showed the same evidence of marked local injury. The length of treatment did not alter the con-

dition, the destruction being as great in the animals treated ten days as in those treated thirty days. Figure 4 shows an anterior section and figure 5 a posterior section.

Treatment with sodium sulfadiazine produced the same picture except that numerous areas of normal tissue were found in all the sections.

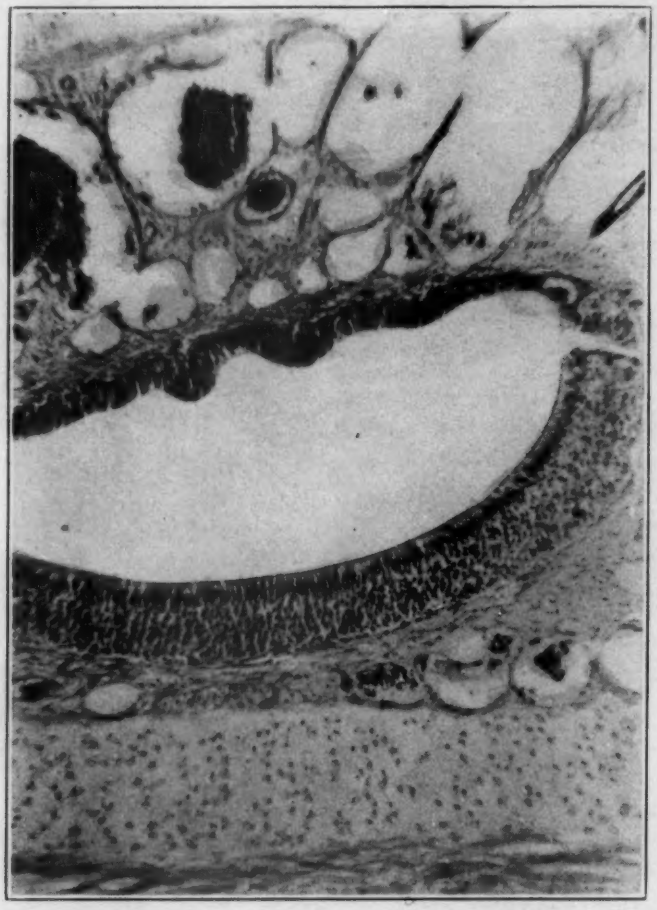

Fig. 6.—Rabbit's nose treated with a solution containing neo-synephrin hydrochloride. Section of a vomeronasal organ from the anterior block. No change from the normal condition is seen.

In a previous experiment, when solutions of sodium sulfathiazole of concentrations up to 25 per cent were instilled into the nose, no trace of the drug could be found in the blood of the heart, indicating that there was very little or no absorption from the nasal mucosa.

No apparent discomfort was caused by the instillation of the solution of neo-synephrin hydrochloride[5a] At autopsy the nasal mucosa in all

the animals treated with this drug remained unchanged. There were no significant variations from the normal mucosa in any of the microscopic sections. Figure 6 shows an anterior section and figure 7 a posterior section.

Sections of the lungs were made in each case. These showed no significant changes in the mucosa of the bronchial tree either in the

Fig. 7.—Rabbit's nose treated with a solution containing neo-synephrin hydrochloride. Anterior tip of a middle turbinate from the posterior block. No change from the normal condition is seen.

animals treated with a sulfanilamide derivative or in those treated with the neo-synephrin hydrochloride solution.[5a]

While our experiments were under way, two apparently conflicting reports on the intranasal use of sodium sulfathiazole appeared in the

literature. Turnbull [8] reported relief of symptoms of chronic sinusitis in 45 of 47 patients treated with a nasal spray of a 5 per cent solution. Fletcher [9] at operation found complete necrosis of the lining membrane of a maxillary sinus into which a 5 per cent and a 10 per cent solution of the same drug had previously been instilled.

COMMENT

Whether the destruction noted when sodium sulfathiazole and sodium sulfadiazine were used was due to the local irritant or toxic action of the drugs or to their high alkalinity, we are not yet in a position to say. However, the fact that the butanoylsulfanilamide solution is so nearly neutral seems to indicate that the destruction of the mucous membranes was caused at least partially by the toxic action of the drugs and not entirely by possible caustic action resulting from the highly alkaline nature of the solutions of sodium sulfathiazole and sodium sulfadiazine.

It is difficult to say definitely how close an application of this work on animals can be made to human beings. Nevertheless, since the intranasal application of the solutions of sulfanilamide derivatives resulted in such severe destruction of the nasal mucosa in rabbits, it seems that one should refrain from using them in the nose until such time as noninjurious solutions of these drugs have been perfected. We are now conducting further experiments with a view to developing a solution that will be noninjurious.

CONCLUSIONS

From these experiments as they apply to the normal nasal mucosa of rabbits we draw the following conclusions:

1. Sodium sulfathiazole and butanoylsulfanilamide in the strength used (4.7 per cent) are locally destructive when used for nasal instillation.

2. Sodium sulfadiazine, while not so completely destructive, is definitely injurious.

3. The vasoconstrictor employed is apparently safe as a nasal medicament when used three times a day and even if used ten times daily for a short period.

Dr. A. R. Camero aided in the study of the microscopic sections, and Dr. E. M. Hall gave the use of his laboratory at the University of Southern California Medical School.

1930 Wilshire Boulevard.

8. Turnbull, F. M.: Intranasal Therapy with Sodium Salt of Sulfathiazole in Chronic Sinusitis, J. A. M. A. **116**:1899-1900 (April 26) 1941.

9. Fletcher, R.: Sodium Sulfathiazole: Its Caustic Action, California & West. Med. **55**:94 (Aug.) 1941

THE SEMICIRCULAR CANALS AS A DEVICE FOR VECTORIAL RESOLUTION

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ABSTRACT

The semicircular canals of the ear are treated from the point of view of the physicist as a device for the resolution of a vector stimulus such as angular acceleration into three mutually perpendicular components, one for each pair of homologous canals. The brain, receiving the nerve impulses from the canals, recombines these components vectorially to measure the angular acceleration to which the individual is subjected. The brain has so evolved that, without consciousness of the intermediate steps required, it yields an immediate answer to this problem whose mathematical solution requires some minutes of tedious computation.

Equations, graphs and a numerical table are given so that the mechanical stimulus to each canal can be found when the head is subjected to angular acceleration about any axis.

It is shown that the sum of the mechanical stimuli to the three pairs of canals is greatest when the angular acceleration is about an axis which makes equal angles with the planes of the canals. The total mechanical stimulus is least when the axis is perpendicular to the plane of any one pair of canals.

PHYSICAL INTRODUCTION

A vector quantity is one, like force or velocity, which has both magnitude and direction. Such a quantity can be represented graphically by a vector, a straight line with an arrowhead at one end. The length of the line indicates the magnitude of the quantity; its orien-

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tation and the arrowhead show the direction of the quantity.[1] As an example, suppose that you hold at rest in your hand a book which weighs 2 pounds (0.9 Kg.) (fig. 1 *a*). Your hand exerts an upward force of 2 pounds on the book. This force may be represented by a vector, a vertical line 2 units long (corresponding to 2 pounds), with an arrowhead at the top to show that the force is upward.

The vector quantities with which we are primarily concerned in this study are linear acceleration, angular velocity and angular acceleration. Linear acceleration is defined as the time rate of change of linear velocity and might be expressed in centimeters per second per second. Angular velocity is the time rate of angular displacement. It may be represented by a vector whose length indicates the time rate of rotation and which lies along the axis about which rotation occurs. The arrowhead points in the way in which the thumb would point if one were to grasp the axis of rotation with the right hand with the fingers around the axis in the direction of rotation (fig. 1 *b*). Angular velocity may be expressed in revolutions per second. If, however, one wants to compute forces or torques which act on a rotating body, one should express angular velocity in radians per second. The radian is an angle which subtends an arc whose length is equal to its radius. There are therefore $\pi(3.1416)$ radians in 1 revolution. In formulas it is customary to use the lower case Greek letter omega (ω) to represent angular velocity. Angular acceleration is the time rate of change of angular velocity. Thus a constant angular acceleration of 3 revolutions per second per second means that in each second the angular velocity increases by 3 revolutions per second. If a body is rotating about a fixed axis, the vector of angular velocity and the vector of angular acceleration are parallel. The conventional symbol for angular acceleration is the lower case Greek letter alpha (α).

COMPONENTS OF A VECTOR

Suppose that a boy pulls a sled, exerting a force F on a cord which is inclined at 25 degrees above the horizontal (fig. 2 *a*). This pull is exactly equivalent to an upward lift $F \sin 25°$ and a horizontal force $F \cos 25°$. F may be called the resultant or the vector sum of these two components. In this illustration we are concerned with vectors which are all in the same plane—coplanar vectors. In applying vector methods to the study of the semicircular canals, we shall have

1. Some physicists prefer to say that a vector has three properties: magnitude, direction and sense. The length of the line indicates the magnitude, the orientation of the line shows the direction and the arrowhead marks the sense. For a given direction there are thus two possible senses, according to the way the arrowhead points.

to deal with vectors which are not coplanar. We shall therefore proceed to a three-dimensional resolution of a vector into components. Let us begin by assuming three mutually perpendicular directions or axes of reference, X, Y and Z (fig. 2 *b*). For convenience, let X and

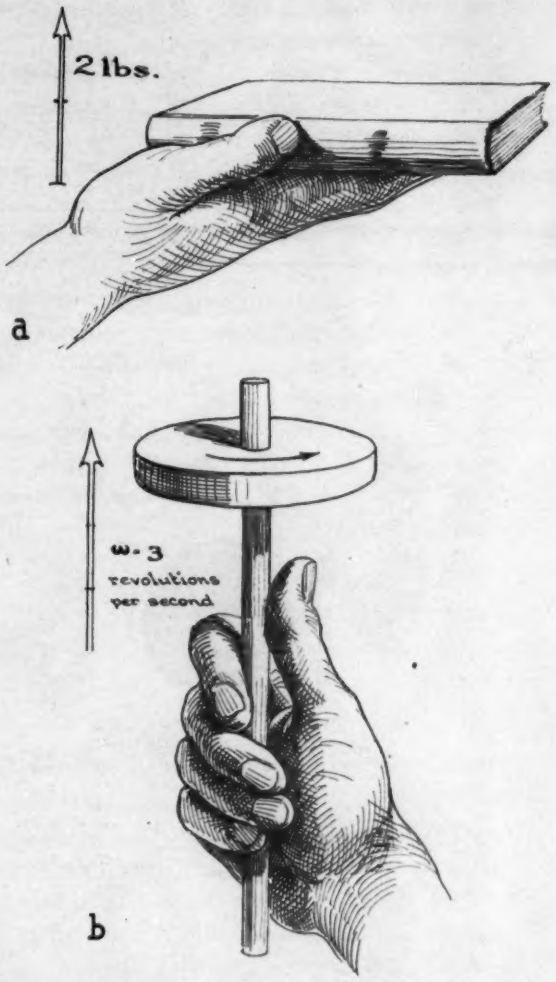

Fig. 1.—(*a*) The vertical arrow is a vector which represents the force exerted by the hand on the book. The length of the vector, 2 units, indicates the magnitude of the force, assumed to be 2 pounds (0.9 Kg.).

(*b*) Angular or rotational velocity, ω, is a vector quantity represented by a vector parallel to the axis of rotation. If the fingers of the right hand are curved around the axis in the direction of rotation, the thumb indicates the sense of the vector, as shown by the arrowhead.

Y be horizontal and Z vertical. As an illustration, consider a top which is spinning at ω revolutions per second about an axis which at

a given instant is at 50° to the horizontal and which lies in a vertical plane that makes an angle of 30° with the X axis, as represented by the line OA (fig. 2 b).[2] To find the Z component of vector ω, draw a line AB from the end of ω perpendicular to the Z axis. This perpendicular cuts off on the Z axis a length $OB = \omega \sin 50°$, which is the

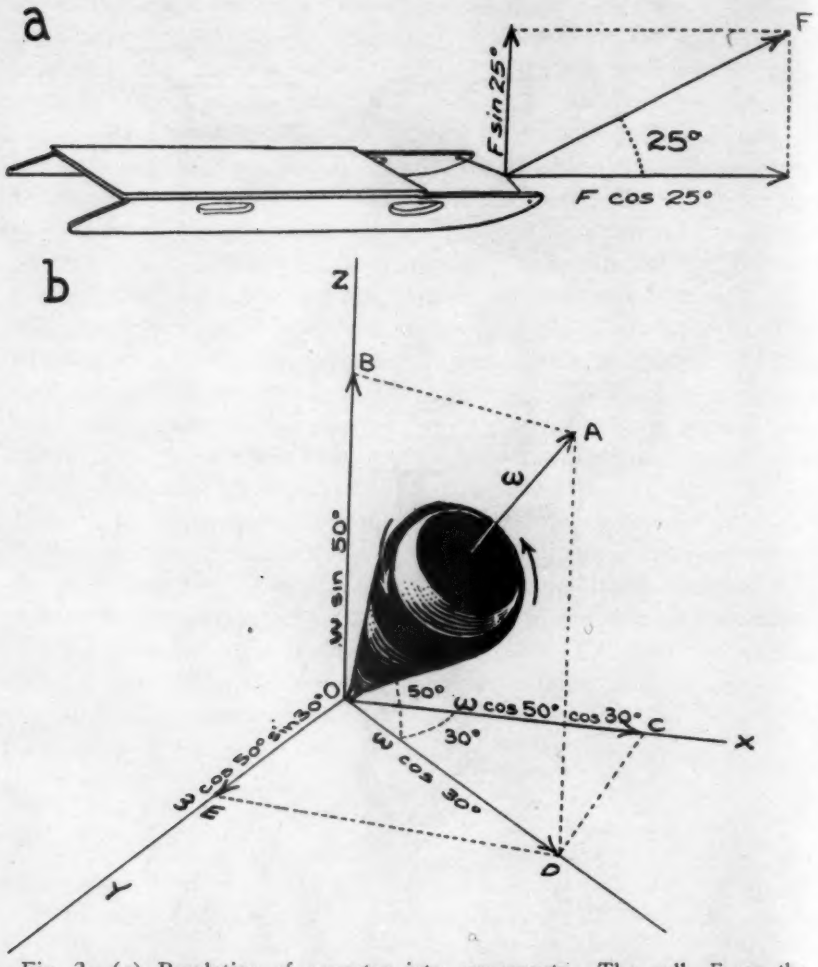

Fig. 2.—(a) Resolution of a vector into components. The pull, F, on the sled, is equivalent to an upward force, F sin 25° and a simultaneous horizontal force, F cos 25°. F is the resultant of these two components.

(b) Three-dimensional example of resolution of a vector quantity, ω, the angular velocity of a spinning top, into components in three mutually perpendicular directions, X, Y, Z.

2. For simplicity, we are considering only the rotation of the top about its own axis and are not concerned with more complex motion, such as precession, which would also be present.

magnitude of the required Z component. Similarly we might drop perpendiculars AC and AE from the end of ω to the X and Y axes to find those components. This, however, is not convenient because the statement of the problem did not specify explicitly the angles between ω and the X and Y axes. We shall therefore use an indirect method. Draw line AD from the end of ω perpendicular to the plane in which lie the X and Y axes. A line OD from the origin O to the point D where the perpendicular AD cuts this plane has a length $\omega \cos 50°$, and this is the magnitude of the component of ω in the XY plane. By drawing perpendiculars DC and DE from the end of this component to the X and Y axes, we find that the X and Y components of ω are respectively $OC = OD \cos 30° = \omega \cos 50° \cos 30°$ and $OE = OD \sin 30° = \omega \cos 50° \sin 30°$. Thus if the top is spinning at ω revolutions per second about its axis, this rotation is equivalent to simultaneous rotations of $\omega \sin 50°$ revolutions per second about a vertical Z axis and $\omega \cos 50° \cos 30°$ and $\omega \cos 50° \sin 30°$ revolutions per second, respectively, about two horizontal axes, X and Y, oriented as illustrated. Conversely, if the X, Y and Z components OC, OE and OB of the vector ω are given, the magnitude, direction and sense of ω are uniquely determined. It is a simple exercise to show that $\sqrt{\overline{OB}^2 + \overline{OC}^2 + \overline{OE}^2} = \sqrt{(\omega \sin 50°)^2 + (\omega \cos 50° \sin 30°)^2 + (\omega \cos 50° \cos 30°)^2} = \omega$. The orientation of ω may be found by the relations $\sin \angle AOD = OB/OA$ and $\sin \angle COD = OE/OD = OE/\left(\sqrt{\overline{OE}^2 + \overline{OC}^2}\right)$, as is evident from figure 2 *b*.

APPLICATION TO THE STUDY OF THE SEMICIRCULAR CANALS

In the haze of uncertainty which still veils the functions of many parts of the bony labyrinth there seems to be general agreement that angular acceleration is a stimulus which affects the semicircular canals. In another paper [3] we show by application of mechanical principles that the form of the canals is such that they might conceivably respond to three types of mechanical stimuli, namely (1) angular acceleration, (2) angular velocity and (3) linear acceleration. These stimuli are all vector quantities.

The present analysis is primarily in terms of angular acceleration. It should be understood, however, that the same geometric analysis is valid for any vector quantity and is hence applicable to any of the aforementioned stimuli. In the case of angular acceleration or angular

3. Morgan, R.; Summers, R. D., and Reimann, S. P.: Effects of Various Types of Motion on Differences in Hydrostatic Pressure Between Ends of a Semicircular Canal: A Theoretic Analysis, Arch. Otolaryng. **36**:691 (Nov.) 1942.

velocity the effective component vector is normal to the plane of a given canal; in the case of linear acceleration the effective component is in the direction of a line joining the two ends of the canal. This difference must be taken into account in applying the results to linear acceleration.

If a physicist were to undertake to measure the magnitude and the direction of the angular acceleration of an object which is subject to such acceleration about any axis whatsoever, he would set up three instruments, one to measure the angular acceleration of the object about each of three mutually perpendicular axes. The readings of the three instruments would correspond to the components of the required angular acceleration about the respective axes of the instruments. From these three components, the magnitude and the direction of the angular acceleration of the object could be computed. This seems like an indirect and artificial procedure, a clumsy method which must be used for want of a better one. The physicist is therefore struck by the fact that the semicircular canals constitute just such an array of three instruments as we have described, each homologous pair of canals serving to measure the magnitude of the angular acceleration of the head about an axis perpendicular to the plane of these canals. Fortunately, the compounding of these three components of angular acceleration to find the resultant occurs below the level of consciousness, so that the subject is aware only of the answer of the problem and spared recourse to mathematics to perceive how his motion is changing. He feels only the resultant angular acceleration of sensible magnitude about an axis which is oriented in a certain direction with respect to himself. A mathematical solution of this vectorial composition would require perhaps fifteen minutes of tedious computation. The use of a computing machine would materially reduce this time but, nevertheless, seven settings of the machine would be necessary. On the other hand, the brain has so evolved that one feels almost instantly the magnitude, direction and sense of the resultant angular acceleration. It would certainly be possible to make a special machine to analyze an acceleration into its components and to recombine them into a resultant described by numbers on suitable dials. Such a machine would be quicker than the brain and would yield results of higher precision, but it would be useful for the solution of that particular type of problem and for nothing else, in marked contrast with the versatility of the brain.

As an introduction to the application of vector methods to the study of the semicircular canals, we shall solve the following problem: The head is subject to an angular acceleration about an axis which is oriented in any given direction; find the relative magnitude of the angular acceleration stimulus on each canal, i. e., the magnitude of

the component of the angular acceleration parallel to the axis of each canal. In other words, a vector quantity is given with reference to a set of coordinate axes, *X, Y, Z*. What are its components in the directions of another set of coordinate axes, *1, 2, 3,* which are normal to the respective planes of the semicircular canals? To simplify the mathematics, we shall limit the present treatment to the case in which the angles between the *X, Y, Z* axes and the *1, 2, 3* axes have instantaneously certain given numerical values. However, the method will be one which can be readily extended to more general cases in which axes *1, 2, 3* have a specified angular motion with respect to axes *X, Y, Z*. We shall accordingly assume a definite instantaneous orientation of the *X, Y, Z* axes with respect to the head (fig. 3):

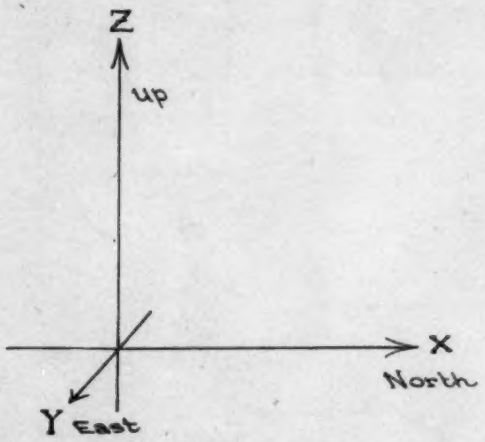

Fig. 3.—Orientation of coordinate axes.

Let the head be in the normal upright position, facing south.

Take the *Z* axis vertical, positive sense upward.

Take the *Y* axis horizontal, west to east, positive sense toward the east.

Take the *X* axis horizontal, south to north, positive sense toward the north.

The geographic directions named are introduced solely to facilitate description and have no further significance. In the present problem, the head may be oriented in any way, provided that the *X, Y, Z* axes are instantaneously fixed with respect to the head in the manner just defined.

To lay a foundation for more advanced problems, we shall introduce Euler's angles[4] to relate the axes of the canals with respect to

4. Leonhard Euler was a Swiss mathematician (1707-1783).

the *X, Y, Z* axes. Imagine the axes *1, 2, 3* initially coincident with the *X, Y, Z* axes, respectively (fig. 4*a*). Displace the axes *1, 2, 3* as a rigid frame in the following manner:

I. Rotation through an angle ψ about the *Z* axis, carrying axis *1* from *OA* to *OA'* and axis *2* from *OB* to *OB'* (fig. 4*b*).

II. Rotation through an angle θ about *OB'* (axis *2*), carrying axis *1* from *OA'* to *OA''* and axis *3* from *OC* to *OC'* (fig. 4*c*).

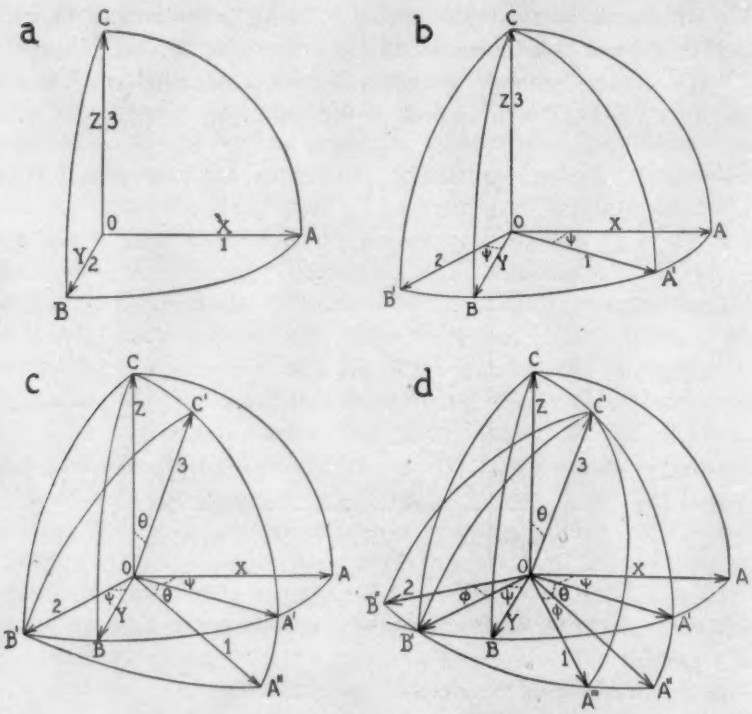

Fig. 4.—Definition of Euler's angles: (*a*) Two sets of coordinate axes, *X, Y, Z* and *1, 2, 3* are coincident. (*b*) Axes *1, 2, 3* are rotated like a rigid frame through angle ψ about *OC*. (*c*) Axes *1, 2, 3* are next rotated through angle θ about *OB'*. (*d*) Axes *1, 2, 3* are finally rotated through angle ϕ about *OC'*.

III. Rotation through an angle ϕ about *OC'* (axis *3*), carrying axis *1* from *OA''* to *OA'''* and axis *2* from *OB'* to *OB''* (fig. 4*d*).

To simplify the discussion, we shall consider the left set of canals. Extension of the results to the right set is simple and obvious. We shall take axis *1* normal to the plane of the left anterior semicircular canal, axis *2* normal to the plane of the left posterior canal and axis *3* normal to the plane of the left lateral canal. Angular acceleration components a_1, a_2, a_3 about these axes will thus correspond to

the respective angular acceleration stimuli to the left anterior, posterior and lateral canals (fig. 5).

Our problem may be restated thus: Given an angular acceleration a with components a_x, a_y, a_z in the respective directions of the X, Y, Z axes; to find the components a_1, a_2, a_3 of the given acceleration in the respective directions of axes *1, 2, 3*. It is possible to write the relations between these components directly from inspection. The work will, however, be more readily comprehensible to those who are unaccustomed to vectors if we divide the derivation into two parts. First we shall express the angular accelerations a_x, a_y, a_z in terms of

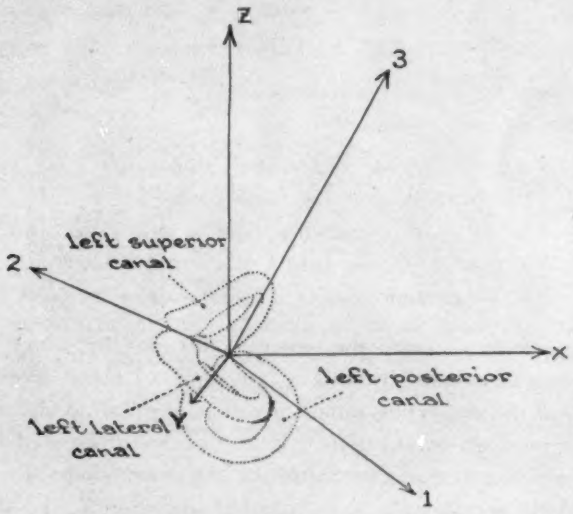

Fig. 5.—Axes *1, 2, 3* are so oriented that each is perpendicular to the plane of a semicircular canal.

a_ψ, a_θ, a_ϕ, the component accelerations about the axes OC, OB' and OC' respectively (fig. 4d):

$$\left.\begin{aligned} a_x &= -a_\theta \sin\psi + a_\phi \sin\theta \cos\psi \\ a_y &= a_\theta \cos\psi + a_\phi \sin\theta \sin\psi \\ a_z &= a_\phi \cos\theta + a_\psi \end{aligned}\right\} \quad (1)$$

The first of the foregoing relations is written in the following way: a_θ is an acceleration about the axis of θ which is OB' (fig. 4*d*). a_θ is therefore represented by a vector in the direction OB'. The angle between OB' and the X axis is $\angle B'OA$ or $90° + \psi$. The X component of a_θ is therefore $a_\theta \cos(90° + \psi)$ or $-a_\theta \sin\psi$, the first term on the right hand side of the equation. Component a_ϕ is an acceleration about the axis of ϕ which is OC'. The angle between OC' and the XY plane is $\angle C'OA'$ or $90° - \theta$. The component of a_ϕ in the XY

plane is $a_\phi \cos(90° - \theta)$ or $a_\phi \sin\theta$ and is in the direction OA'. This component must again be resolved to find the X component. The angle between OA' and the X axis is $\angle A'OA$ or ψ. The X component of $a_\phi \sin\theta$ (and of a_ϕ) is therefore $a_\phi \sin\theta \cos\psi$, the second term on the right hand side of the equation. a_ψ is an acceleration about OC, the axis of ψ. This axis is perpendicular to OA, the X axis, so that a_ψ has no component along the X axis. Our first equation is therefore complete. The equations for a_y and a_z are written by a similar process.

Similarly we may write equations for a_1, a_2, a_3 in terms of a_θ, a_ϕ, a_ψ:

$$\left.\begin{aligned} a_1 &= a_\theta \sin\phi - a_\psi \sin\theta \cos\phi \\ a_2 &= a_\theta \cos\phi + a_\psi \sin\theta \sin\phi \\ a_3 &= a_\psi \cos\theta + a_\phi \end{aligned}\right\} \quad (2)$$

If now by algebra we eliminate a_θ, a_ψ and a_ϕ between equations (1) and (2) we obtain

$$(3)\quad \left\{\begin{aligned} a_1 &= {}_x(\cos\phi \cos\psi \cos\theta - \sin\phi \sin\psi) + a_y(\cos\phi \sin\psi \cos\theta + \sin\phi \cos\psi) - a_z \sin\theta \cos\phi. \\ a_2 &= -a_x(\sin\phi \cos\psi \cos\theta + \sin\psi \cos\phi) + a_y(-\sin\phi \sin\psi \cos\theta + \cos\phi \cos\psi) + a_z \sin\theta \sin\phi. \\ a_3 &= a_x \sin\theta \cos\psi + a_y \sin\theta \sin\psi + a_z \cos\theta. \end{aligned}\right.$$

Equations (3) are the general equations by which we may obtain the component accelerations a_1, a_2 and a_3 about axes normal to the planes of the respective canals, given an angular acceleration a impressed on the head about any axis. It should be emphasized that these equations yield instantaneous values of the components a_1 a_2, a_3. If the impressed acceleration a is constant and the X, Y, Z axes are held fixed in space as the head moves, the components a_θ, a_ψ, a_ϕ are continually changing. However, in many cases, including the important instance of rotation about a fixed axis, the angular acceleration and the angular velocity are about the same axis; as the head turns, the *1, 2, 3* axes rotate as a rigid frame about that axis and make constant angles with it. The magnitudes of a_1, a_2 and a_3 therefore remain constant fractions of the magnitude of a, even though θ, ψ and ϕ may be changing. In such motion, the angular acceleration stimuli to the respective canals will remain constant if the magnitude of a is constant.

In the most general case, angular velocity and angular acceleration are not about the same axes. Then equations (3) are strictly valid only when the head is instantaneously in the position which corresponds to the particular numerical values which we substitute in the equations. In actual practice, however, the possible precision is low because we do not know exactly the orientation of the canals in the head of a particular subject. We are therefore justified in using

the results of equations (3) as the head moves through small angles more or less than the angular values substituted in the equations. The amount of this angular tolerance can be determined only by experience and by careful analysis of the precision required. As the equations are certainly valid only for rotation about a fixed axis, great caution should be used in all other cases.

To illustrate the application of our formulas, let us examine a simple case. The head in a normal upright position is subjected to an angular acceleration about a horizontal axis which makes any angle, β, with the intermeatal line. By analogy to astronomic usage, it is appropriate to call angle β the azimuth of the acceleration a, whose X, Y, Z components will be $a_x = a \sin \beta$, $a_y = a \cos \beta$, $a_z = O$, in which β is assigned values from 0 to 360°. The orientations of the respective canals may be described by $\psi = O, \theta = 30°, \phi = 45°$ (fig. 6).[5] Equations (3) thus become:

$$\left.\begin{aligned} a_1 &= a\ (\sin \beta \cos 45° \cos 30° + \cos \beta \sin 45°) \\ &= a\ [(-\sqrt{6} \sin \beta)/4 + (\sqrt{2} \cos \beta)/2] \\ a_2 &= a\ (-\sin \beta \sin 45° \cos 30° + \cos \beta \cos 45°) \\ &= a\ [(\sqrt{6} \sin \beta)/4 + (\sqrt{2} \cos \beta)/2] \\ a_3 &= a \sin \beta \sin 30° = a \sin \beta/2. \end{aligned}\right\} \quad (4)$$

To facilitate interpretation and plotting of these equations, let us assume a new constant, A, and an angle, γ, defined by the relations

$$\left.\begin{aligned} A \sin \gamma &= \sqrt{2}/2 \\ A \cos \gamma &= \sqrt{6}/4. \end{aligned}\right\} \quad (4a)$$

Dividing the first of these equations by the second we obtain

$$\tan \gamma = 2/\sqrt{3},\ \gamma = 49°6'$$

Adding the squares of the same equations, we obtain $A^2 = 7/8$, $A = 0.935$.

Substitution of equations (4a) into equations (4) yields

$$\left.\begin{aligned} a_1 &= aA\ (\sin \beta \cos \gamma + \cos \beta \sin \gamma) = aA \sin (\gamma + \beta) \\ a_2 &= aA\ (-\sin \beta \cos \gamma + \cos \beta \sin \gamma) = aA \sin (\gamma - \beta) \\ a_3 &= (a \sin \beta)/2 \end{aligned}\right\} \quad (5)$$

These equations are most easily plotted in polar coordinates (fig. 7). In each figure it is evident from the equations that POQ is a right triangle. As vertex P of each right angle lies on the curve, each curve must be a circle, for the locus of the vertex of a right triangle having a given hypotenuse as base is the circumference having the same hypotenuse as diameter. In each diagram of figure 7, the magnitude of a

5. These values correspond with the average orientations of the canals as stated by F. W. Watkyn-Thomas and A. L. Yates (Principles and Practice of Otology, London, H. K. Lewis & Co., Ltd., 1932).

respective component a_1, a_2 or a_3 is represented by the length of the radius vector, OP, drawn from the origin to the circumference of the circle at the given angle of azimuth β. For values of β between 0 and 180° we have in each case one circle; for values between 180° and 360° we have a second circle touching the first circle. Therefore each of equations (5) may be represented in polar coordinates by a pair of equal osculating circles.

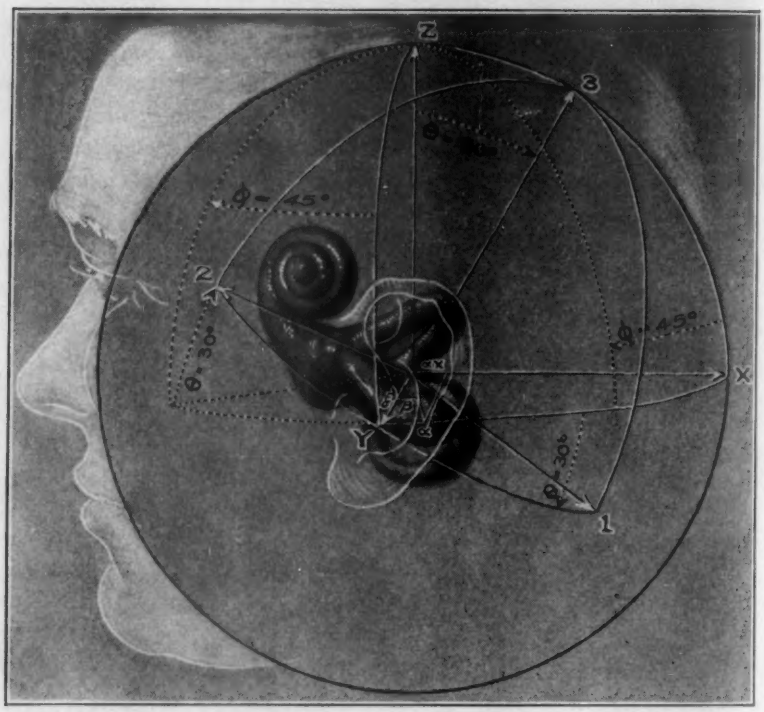

Fig. 6.—To illustrate an application of the derived formulas (equations [3]), numerical values of ψ, θ and ϕ are assumed, as indicated in this figure, together with the corresponding orientation of the left set of canals. No attempt has been made to show the correct size or the location of the canals in head. As $\psi = 0$, it does not appear in the figure.

Let us now investigate the significance of the quantities A and γ. It is evident from figure 7 and equations (5) that αA is the maximum stimulus to an anterior or a posterior canal when the head is subjected to an angular acceleration α about a horizontal axis. A is therefore the cosine of the angle between the normal axis of each of these canals and the XY (horizontal) plane. γ is the angle between the X axis and the projection of the XY plane of the normal axis of an anterior or a posterior canal.

We shall now examine a more general case in which the head in a normal upright position is subjected to angular acceleration about an axis in any direction. Again let α represent the impressed acceleration which lies in a vertical plane whose azimuth or angle with the intermeatal line is β. In this vertical plane, α makes an angle δ, called the altitude, above the (horizontal) XY plane. With β ranging from 0 to 360°, all possible orientations may be had if we restrict δ to values between $-90°$ and $+90°$. The X, Y, Z components of α are

$$\left.\begin{aligned} \alpha_x &= \alpha \cos \delta \sin \beta \\ \alpha_y &= \alpha \cos \delta \cos \beta \\ \alpha_z &= \alpha \sin \delta \end{aligned}\right\} \quad (6)$$

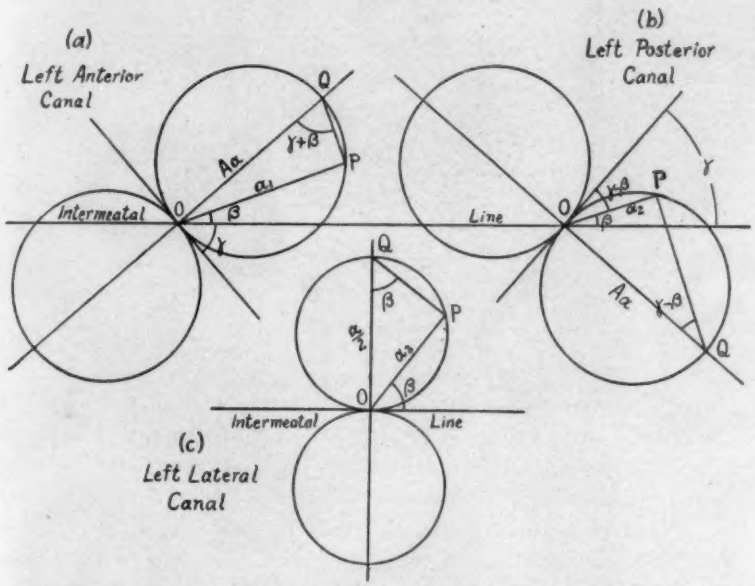

Fig. 7.—Graphic construction for the interpretation of equations (5). If the head is subjected to an angular acceleration α about a horizontal axis which makes an angle β with the intermeatal line, the component stimuli to the canals are represented by the lengths of lines OP in the respective diagrams above. The loci of P are pairs of osculating circles.

As in the preceding case, we shall assume $\psi = O$, $\theta = 30°$, $\phi = 45°$. Equations (3) now become

$$\left.\begin{aligned} \alpha_1 &= \alpha\ (\cos \delta \sin \beta \cos 45° \cos 30° + \cos \delta \cos \beta \\ &\quad \sin 45° - \sin \delta \sin 30° \cos 45°) \\ \alpha_2 &= \alpha\ (-\cos \delta \sin \beta \sin 45° \cos 30° + \cos \delta \cos \beta \\ &\quad \cos 45° + \sin \delta \sin 30° \sin 45°) \\ \alpha_3 &= \alpha\ (\cos \delta \sin \beta \sin 30° + \sin \delta \cos 30°). \end{aligned}\right\} \quad (7)$$

These equations may be simplified by steps analogous to those used in reducing equations (4) to equations (5). In addition to A and γ, defined by equations (4a), we shall introduce the symbols B, C, D, ϵ, η, ν, which are defined by equations

$$\left.\begin{aligned} B \sin \epsilon &= A \sin (\gamma + \beta) \\ B \cos \epsilon &= \sqrt{2}/4 \\ C \sin \eta &= A \sin (\gamma - \beta) \\ C \cos \eta &= \sqrt{2}/4 \\ D \sin \nu &= (\sin \beta)/2 \\ D \cos \nu &= \sqrt{3}/2 \end{aligned}\right\} \quad (7a)$$

Solving equations (7a) by the same method used for equations (4a), we obtain

$$\left.\begin{aligned} B &= [A^2 \sin^2 (\gamma + \beta) + \tfrac{1}{8}]^{\frac{1}{2}} \\ C &= [A^2 \sin^2 (\gamma - \beta) + \tfrac{1}{8}]^{\frac{1}{2}} \\ D &= (\sin^2 \beta + 3)^{\frac{1}{2}}/2 \\ \tan \epsilon &= \sqrt{7} \sin (\gamma + \beta) \\ \tan \eta &= \sqrt{7} \sin (\gamma - \beta) \\ \tan \nu &= (\sin \beta)/\sqrt{3} \end{aligned}\right\} \quad (7b)$$

These symbols being used, equations (7) reduce to

$$\begin{aligned} a_1 &= aB \sin (\epsilon - \delta) && \text{(a)} \\ a_2 &= aC \sin (\eta + \delta) && (8)\ \text{(b)} \\ a_3 &= aD \sin (\nu + \delta). && \text{(c)} \end{aligned}$$

These equations may be represented by three dimensional graphs, i. e., by curved surfaces in space. If we hold constant ϵ, which depends on the single variable β, equations (8a) may be considered to be the equation of the line of intersection of the surface of equation (8a) and a vertical plane of azimuth β. This line of intersection is a pair of circles, for the form of equation (8a) with ϵ constant is the same as the form of one of equations (5). Figure 8 is a perspective drawing showing some of these pairs of circles (*i, ii, iii*) which correspond to different values of β. The diameter of each circle is the value of aB which corresponds to the given value of β. Circles *iv* are in a horizontal plane corresponding to $\delta = 0$ and are the same circles illustrated in figure 7 *a*. All of these circles lie on the surfaces of a pair of osculating spheres represented by the two large circles *v* in figure 8. Equation (8a) is the equation of this pair of spheres. Similarly, equations (8b) and (8c) represent pairs of spheres.

To learn more about these spheres, let us examine the simplest case, that of equation (8c). As $D = (\sin^2 \beta + 3)^{\frac{1}{2}}/2$, D will have its maximum value when $\beta = 90°$ or $270°$. Then $D_{max} = (1 + 3)^{\frac{1}{2}}/2 = 1$. The diameter of each of these spheres will therefore equal the magnitude of the impressed acceleration a. When D has this maximum value,

tan $\nu = (\sin \beta)/\sqrt{3} = \pm 1/\sqrt{3}$ and $\nu = 30°$ or $210°$ if $\beta = 90°$ or $\nu = 150°$ or $330°$ if $\beta = 270°$. When the magnitude of a_3 has its greatest value (a), sin $(\nu + \delta) = \pm 1$. Therefore $(\nu + \delta) = 90°$ or $270°$. All possible combinations of these values of ν and of $(\nu + \delta)$ lead to two sets of values of δ and β: $\delta = 60°$, $\beta = 90°$ and $\delta = -60°$, $\beta = -90°$. Both of these sets of results describe the same line which is normal to the plane of the lateral canal. In other words, the angular acceleration stimulus to the lateral canal is greatest when the total applied angular acceleration is about an axis which is normal to the

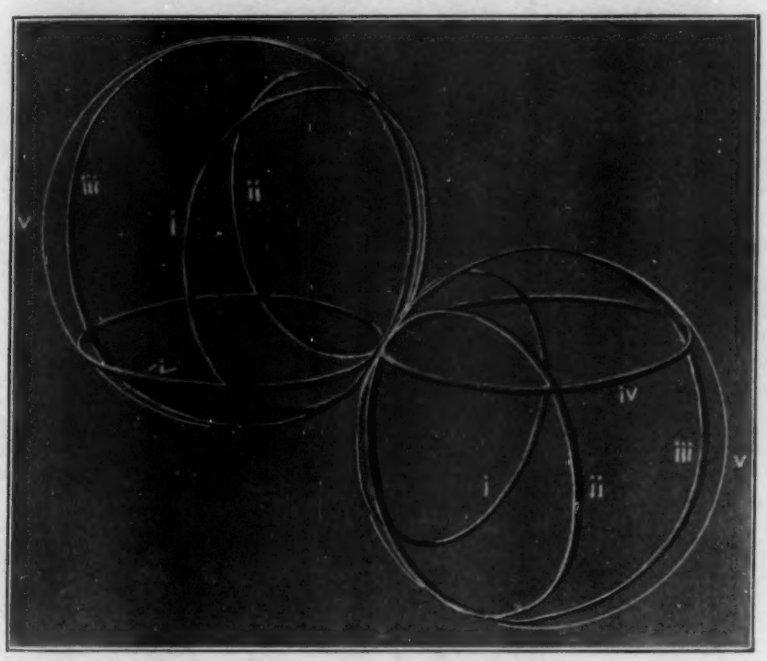

Fig. 8.—Graphic construction for the interpretation of equations (7) and (8). The head is subjected to angular acceleration, a, about an axis oriented in any way. Spheres v, of diameter A, have their common diametral line perpendicular to the plane of a canal. The mechanical stimulus to the canal is represented by the length of a line in the direction of the axis of the acceleration drawn from the point of contact of the two spheres until it meets the surface of the spheres. Circles *i*, *ii*, *iii* and *iv* lie on the surface of the spheres, and each is the locus of the end of such a line in a particular case. Horizontal circles *iv* correspond to circles in figure 7 *a*.

plane of that canal. We already knew this fact, so that its deduction serves as a partial check on the mathematics. From another point of view, we have shown that the common diametral line of the twin spheres is normal to the plane of the lateral canal. By similar analysis

of equations (8a) and (8b) analogous conclusions can be obtained for the spheres associated with the anterior and posterior canals.

We may summarize these results by the following graphic rule: To find the stimulus to a given semicircular canal when a given angular acceleration is impressed on the head, imagine two equal spheres in contact with each other, their common diametral line in the direction normal to the plane of the given canal. Let the diameter of each sphere equal the magnitude of the total angular acceleration. Imagine a straight

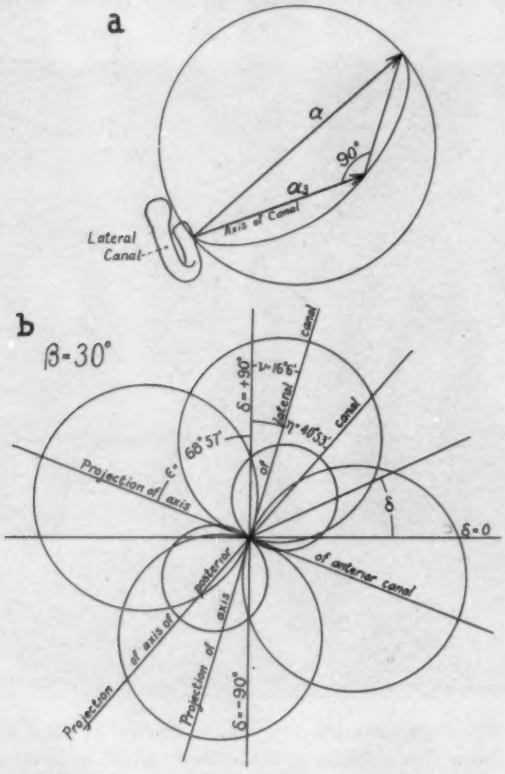

Fig. 9.—(*a*) Alternative graphic construction. Construct a sphere whose diameter is the angular acceleration vector α. A chord of this sphere from the origin of vector α, perpendicular to the plane of any canal, will represent the angular acceleration component to which this canal responds.

(*b*) Auxiliary graph for the determination of the component angular acceleration in the direction of the axis of each canal, when the resultant angular acceleration vector lies in a vertical plane which is at angle $\beta = 30°$ with the intermeatal line. Three diagrams of the type shown in figure 7 are here superposed.

line through the point of contact of the spheres and parallel to the axis of the acceleration, i. e., parallel to the vector of the angular acceleration. The length of this line from the point of contact of the spheres to the

surface of either sphere will represent the magnitude of the required angular acceleration stimulus. The sense of the angular acceleration stimulus is obvious; however, the following rule may be stated: Consider the point of contact of the spheres as the origin of vectors. The sense of the component angular acceleration will be that of the diametral vector drawn from the origin in that sphere which is pierced by the total angular acceleration vector, also drawn from the origin.

The spheres which we have drawn enable us to picture the component stimulus to any canal when the angular acceleration is about any axis. Another construction may be useful, to picture the component stimulus to a canal when the *canal* is oriented in any way. Draw a vector to represent the angular acceleration and imagine a sphere of which this vector is a diameter. Through the end of the vector diameter at its origin draw a chord of the sphere parallel to the normal axis of a semi-

Numerical Values Calculated from Equations (7 b), for Use in Plotting the Three-Dimensional Graph, Figure 11

β	B	C	D	ϵ	η	ν
0°	0.792	0.792	0.866	63°26′	63°26′	0°00′
10°	0.877	0.688	0.870	66°14′	59°04′	5°43′
20°	0.943	0.577	0.884	67°58′	52°09′	11°23′
30°	0.985	0.468	0.902	68°57′	40°53′	16°6′
40°	1.000	0.384	0.925	69°18′	22°42′	20°21′
50°	0.990	0.354	0.948	69°03′	— 2°23′	23°50′
60°	0.951	0.396	0.970	68°12′	—26°35′	26°35′
70°	0.890	0.487	0.986	66°37′	—43°21′	28°27′
80°	0.808	0.597	0.998	64°02′	—53°39′	29°35′
90°	0.707	0.708	1.000	60°00′	—60°00′	30°00′

circular canal. This chord, considered as a vector from the origin, represents the stimulus to the given canal (fig. 9 *a*).

As there is no very satisfactory way to plot graphs in three dimensions, it may be necessary to use the equations to compute quantitative results. The accompanying table, relating to our next problem, may be useful in such computation.

As a final exercise, we shall solve this problem: Find the sum of the magnitudes of the stimuli to all the canals when the head is subjected to angular acceleration about any axis. In other words, find $|a_1|+|a_2|+|a_3|$ when each pair of vertical bars means that we shall consider only the magnitude of the enclosed quantity and treat it as positive.

Outline of solution:

I. Compute by equations (7b) the numerical values of B, C, D, ϵ, η, ν for $\beta = 0°$, 10°, 20°, 30°, 40°, 50°, 60°, 70°, 80°, and 90°. See the table.

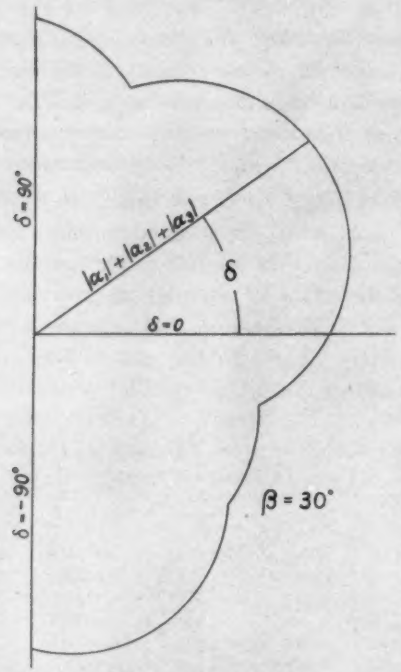

Fig. 10.—The sums of the magnitudes of the three component angular accelerations, found by graph (fig. 9*b*), are plotted against δ, the inclination of the resultant acceleration above the horizontal.

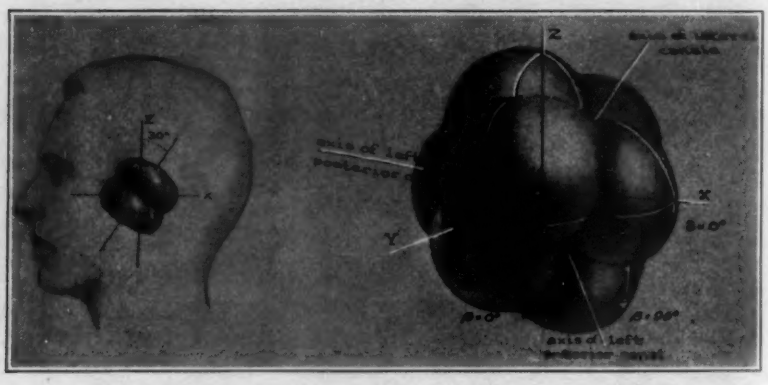

Fig. 11.—Graphs like figure 10, at 10° intervals of β, grouped about a common vertical (Z) axis, determine a three-dimensional surface which is found to be a symmetric arrangement of parts of eight spheres. The sum of the magnitudes of the mechanical stimuli to the canals is represented by the length of a line in the direction of the axis of angular acceleration from the origin O to the surface of the figure.

II. Assume a convenient length such as 10 cm., to represent the magnitude of a. On a separate graph for each value of β plot the six circles, i. e., a pair each for a_1, a_2, a_3. Figure 9 *b* shows a specimen graph for $\beta = 30°$. The coordinates are δ and a_1, a_2, a_3.

III. Using as radii vectores values of $|a_1|+|a_2|+|a_3|$ measured on the aforementioned graphs, plot for values of δ from $-90°$ to $+90°$ ten more graphs, one for each value of β. Figure 10 shows one of these graphs and figure 11 is a perspective drawing of the ten graphs assembled at appropriate values of β.

It is unnecessary to plot graphs as in figure 9 *b* for values of β between 90° and 360° because the resulting three-dimensional graph (fig. 11) is symmetric about the sagittal plane ($\beta = 90°$) and also about the origin of coordinates.

Figure 11 reveals two interesting facts which might have been predicted from general properties of vectors and which can be readily verified by application of calculus to equations (4):

1. The sum of the stimuli to all the canals is greatest when the axis of the angular acceleration makes equal angles with the normal axes of all the canals—i. e., when the stimuli to all the canals are equal. There are four such axes for maximum total stimulus. In figure 11 these maxima appear at $\delta = 30°$, $\beta = 19°30'$; $\delta = 5°16'$, $\beta = 90°$ and at $\delta = -65°16'$, $\beta = 90°$, etc.

2. The sum of the stimuli is least when the axis of the angular acceleration is parallel to the normal axes of any one pair of homologous canals, so that only that pair of canals is affected. There are three such axes of minimum stimulus. In figure 11, such minima appear at $\delta = -20°42'$, $\beta = 40°54'$ when only the left posterior and right anterior canals are affected and at $\delta = 60°$, $\beta = 90°$, when only the lateral canals are affected, and at $\delta = 20°42'$, $\beta = -40°54'$ for the left anterior and right posterior canals.

Case Reports

ANTHRAX APPEARING PRIMARILY IN THE NOSE

FREDERICK KRAUSS, M.D., PHILADELPHIA

The interesting feature of this case is infection with the anthrax bacillus, apparently by means of a fine dust in a factory making leather art goods. The dust inhaled through the nose caused primarily a severe allergic type of swelling of the turbinates and mucous membranes and secondarily an intense swelling covering the whole neck and chest. No evidence of the organism could be found until thirty-six hours after admission of the patient to the hospital.

REPORT OF A CASE

W. S., 71 years of age, was well until May 4, 1941, when he began to feel very tired. The following day he noticed a dryness in his throat and nose, followed by headache with running of the nose. That same evening he noticed a fulness on the right side of his neck but had no pain or tenderness. On May 6 he had attacks of difficulty in breathing and swallowing. That night he was admitted to the Abington Hospital.

History.—He had had the usual childhood diseases. In 1921 an appendectomy was performed, and in 1936 he had pneumonia. His father died of "throat consumption."

The patient was a master carpenter and mechanic engaged in construction and repair work in an art leather factory where leather was brushed with sandpaper. There was much dust flying around.

Physical Examination.—At 9 p. m. his temperature was 100.8 F., his pulse rate 100 and his respiratory rate 20. The blood pressure was 134 systolic and 94 diastolic. He appeared to be in great distress, breathing rapidly through the mouth, but seemed fairly alert. There was noticeable lacrimation without inflammation of the eyes. The nasal passages were completely blocked on both sides, with intense pallor of the mucous membranes covering the swollen turbinates, such as is seen in extreme forms of allergy. The neck showed a diffuse swelling, but no tenderness and no induration. This swelling occupied the whole area between the lower part of the face and the clavicle on both sides of the neck and was rather more pronounced on the right side.

Repeated packing with a 1 to 1,000 solution of epinephrine hydrochloride and application of cocaine solutions were without effect. Forced packing with tampons caused no bleeding. The pharynx and larynx showed no apparent lesion, and there was no edema or obstruction in the larynx. Smears and cultures of material from the nose and pharynx, as well as a culture of the blood, were made at once.

Laboratory Report.—The hemoglobin content of the blood was 104 per cent (14.88 Gm.). There were 5,330,000 red blood cells and 10,700 white blood cells per cubic millimeter. Eighty per cent of the white cells were polymorphonuclears, 16 per cent lymphocytes, 3 per cent monocytes and 1 per cent basophils. There were 120,000 platelets. The results of the Wassermann and Kahn tests were

From the Department of Otolaryngology, Abington Memorial Hospital, Abington, Pa.

negative. The urea nitrogen content of the blood was 18 mg. per hundred cubic centimeters and the sugar content 105 mg. The urine had a specific gravity of 1.036 and contained a faint trace of albumin. Microscopic examination revealed urates (1 plus), squamous epithelium (2 plus) and 8 to 10 white cells per high power field.

Culture of material taken from the throat on admission showed Streptococcus viridans. Culture of material from the nose showed pneumococci. Nasal secretion obtained twenty-four hours after admission showed a small number of anthrax bacilli in the culture. The blood showed Bacillus anthracis in culture thirty-six hours after being taken.

On the patient's admission 30 grains (1.9 Gm.) of sulfanilamide with sodium citrate was given, and the dose was repeated every four hours during the night. After consultation with Dr. S. A. Cross this medication was discontinued, and sulfathiazole in doses of 15 grains (0.98 Gm.) every four hours was given the following day. We were unable to reduce the swelling of the tissues of the nose for about thirty-six hours. There was no pus or discharge of any kind, simply intense edema. On the night of May 7, after one hour of persistent forcible packing and repacking of the nostrils, a narrow entrance was made opposite the middle turbinates on the left side, where a small amount of mucus was seen and cultured.

In view of the history of the patient's working in a factory filled with dust from leather, we were searching for the spores of anthrax bacilli. On May 8, thirty-six hours after his admission, they were found in the mucus obtained the night before from the left nostril. Shortly afterward the blood culture revealed anthrax bacilli. The swelling of the neck increased enormously, extending over the upper half of the chest, and assumed a bluish black tint. The patient had fainting attacks, becoming pulseless for thirty seconds. Immediately on discovery of the anthrax bacilli he was given 300 cc. of antianthrax serum followed by 1,000 cc. of 5 per cent solution of dextrose in distilled water by hypodermoclysis and 1,000 cc. of physiologic solution of sodium chloride intravenously. Later he was given an additional 300 cc. of antianthrax serum followed by 1,000 mg. of ascorbic acid. No reaction followed the injection of the serum, but swelling of the face, neck and chest increased steadily. Breathing was frequently interrupted. The daily output of urine was reduced to 300 cc. The pulse and blood pressure were impossible to take.

On May 10, 300 cc. of antianthrax serum followed by 1,000 cc. of a 5 per cent solution of dextrose in physiologic solution of sodium chloride was given. One-thirtieth grain (2 mg.) of strychnine sulfate was given hypodermically in repeated doses. Atropine, epinephrine and caffeine proved of no avail, and the patient died at 6:05 p. m.

On the day previous to his death the nasal mucous membranes and turbinates lost their swelling spontaneously, which allowed free breathing through the nose. No local ulcer could be found in the nose or pharynx.

Autopsy.—The body was that of an unembalmed 71 year old white man, measuring 66 inches (168 cm.) in length and weighing approximately 160 pounds (72.5 Kg.). Postmortem rigidity was pronounced; postmortem lividity, absent. On external inspection there was a striking degree of swelling of the neck and superior portions of the thorax, so that the normal neck line was obliterated. The swelling extended down to the level of the xiphoid process. The skin over this area presented a patchy reddish purple to red discoloration.

The body was opened with a Y-shaped incision. When the tissues over the thorax were cut and the tissues over the neck were reflected, they were found to be markedly edematous and hemorrhagic. The muscles of the neck were pale red and swollen. The normal relationship of the abdominal viscera had been preserved. The peritoneum was smooth and glistening. In the thorax approximately 300 cc. of brown-tinged fluid was present in the left pleural cavity and 700 cc. in the right pleural cavity. The tissues of the mediastinum were involved in the process seen in the superficial tissues of the thorax. They were edematous and hemorrhagic. The superior vena cava was considerably distended. When the pericardium was opened the heart in situ was found to be of normal size. The sac contained about 100 cc. of straw-colored fluid. The mediastinum and retroperitoneal lymph nodes were extremely hemorrhagic. This condition was most marked in the lymph nodes of the mesentery of the small intestine. At a distance of approximately 100 cm. from the ileocecal valve there were small (1 cm.) hemorrhagic areas on the serosa of the ileum. Sectioning of the bowel revealed corresponding hemorrhagic areas over the mucosa.

The heart was of normal size and shape and rather soft in consistency. The subepicardial fat was slightly increased in amount. The surface was smooth and glistening and reddish gray. There was a moderate degree of prominence and tortuosity of the coronary vessels. On section the myocardium was grayish red and regular; the endocardium was reddish gray, smooth and glistening. The valves presented no abnormalities with the exception of a slight degree of sclerosis of the aortic cusps.

The thoracic and abdominal portions of the aorta showed a rather far advanced arteriosclerotic process with a few small ulcerations.

There was bilateral hydrothorax. The lungs were of normal size and shape, with moderately decreased aeration. The surfaces were reddish gray to dark red, being darker in the dependent parts. On section portions of the organs retracted. The cut surface was reddish gray to hemorrhagic throughout; considerable quantities of blood were present.

The kidneys were of normal size and shape. The surfaces were smooth and glistening and reddish gray. The capsule was slightly thickened and on stripping revealed a moderately coarse granular surface. The architecture was fairly distinct. The pelvic fat was slightly increased in amount. Both kidneys showed mild arteriosclerosis. The left kidney contained a small stone lying over one papilla. The stone was readily dislodged. Nearby there was a smaller similar stone adherent to one of the papillae.

The ureters, bladder and prostate appeared normal.

The liver was slightly increased in size. Its surface was smooth and glistening and brownish yellow. Examination of a section showed the architecture to be indistinct. The venous sinuses were prominent, and considerable quantities of blood were present over the cut surface.

The pancreas revealed no abnormalities.

The spleen was of normal size and shape. The capsule was grayish purple and slightly thickened. On section numerous trabeculae lying in a slightly soft, reddish purple pulp were found.

The gastrointestinal tract presented no lesions other than those previously mentioned.

In general, there was evidence of septicemia and toxemia, with edema of the tissues of the neck and mediastinum and hemorrhages of all lymph glands and lymphoid tissues.

Anatomic Diagnosis.—The anatomic diagnosis was as follows: parenchymatous degeneration of the heart with moderate coronary sclerosis and myosclerosis; patchy atelectasis of the lungs, with congestion and edema and bilateral hydrothorax; congestion and chronic passive congestion of the liver, with parenchymatous degeneration; fibrosis and congestion of the spleen; mild arteriosclerosis of both kidneys and lithiasis of the left; in general, edema of the tissues of the neck and mediastinum, hemorrhages of all lymph glands and lymphoid tissues and septicemia and toxemia.

Postmortem Bacteriologic Findings.—A culture of tissue from the spleen showed B. anthracis. A culture of blood from the heart showed B. anthracis and Bacillus coli communis.

The occurrence of anthrax remains a difficult problem for leather manufacturers and leather workers. In spite of many precautions, the spore-laden dust comes in contact with almost any exposed part of the body. One infection is reported to have occurred in the wife of a workman who had worked in such dust. Lucchesi and Gildersleeve [1] in a recent report of cases of anthrax stated that in 17 the lesion was on the forearm, in 12 on the face, in 10 on the neck, in 3 on the chest and in 1 each on the hand, wrist, axilla and back. These lesions were easily recognized, and with early treatment all the patients recovered.

The fatal cases apparently are those in which the spores enter through the mucous membranes, such as those of the nose, pharynx and intestinal tract.

I had 1 case in which the conjunctival surface became infected. There was no visible lesion except an intense edema involving the right side of the face. All efforts to find the offending organism were ineffectual until it was found in the spinal fluid several days later, when meningitis had developed.

I have been unable to find any reference in the literature to a primary involvement of the nasal sinuses. I am much impressed with the intense edema, accounted for by no obvious lesion, which occurs in these cases. There is usually no induration in this edema, which does not pit on pressure and is apparently due to free fluid in the tissues of the neck and in the mediastinum, as was revealed by the autopsy on the patient whose case has been reported here.

1. Lucchesi, P. F., and Gildersleeve, N.: Treatment of Anthrax, J. A. M. A. **116**:1506-1508 (April 5) 1941.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

(Concluded from Page 107)

THERAPEUTIC MEASURES

Hill [37] attempts to evaluate the various forms of therapy for defective hearing. A careful study of the patient to determine etiologic factors and a thorough appraisal of the hearing function will suggest more definite application or limitation of treatment, depending on age and mental or economic status. The author discusses various forms of treatment employed, such as local therapy, irradiation, administration of vitamins, the fenestration operation and employment of hearing aids. Hill feels that radiation therapy should be reserved for those patients whose defect defies the more orthodox procedure of a carefully performed adenoidectomy, and that its employment should be restricted to physicians having both the equipment for adequate dosage and the experience to govern its use. The fenestration operation is still the subject of much controversy. When the extent of the pathologic process cannot be ascertained and the question of further extension of the process cannot be answered, it is difficult to evaluate this operation in terms of permanent effectiveness. Hill suggests that for the majority of patients with irreversible impairment of hearing sufficient to be a handicap, a properly selected hearing aid is the best form of therapy.

Fowler [38] discusses various nonsurgical treatments for deafness. He emphasizes first the importance of the otologist's taking personal interest in his patient. He further adds that an improvement in hearing of less than 15 decibels for the speech range, especially 1024 and 2048 cycles,

37. Hill, F. T.: What Otologists Can Do for Defective Hearing, Tr. Am Acad. Ophth. (1941) **46**:242 (May-June) 1942.

38. Fowler, E. P., Jr.: Nonsurgical Treatment for Deafness, Laryngoscope **52**:204 (March) 1942.

should not be considered significant in view of the variable conditions under which audiograms are made. He has found the treatment with prostigmine methylsulfate unsatisfactory in a series of 90 cases and reports several types of reaction to the drug, such as rash, epigastric distress, diarrhea and headache.

He discusses other forms of therapy, such as injections of tuberculin and insulin, tubal inflations and roentgen and radium therapy to the eustachian tubes and nasopharynx. He has found roentgen therapy effective in improving the patency of the eustachian tubes in cases of stricture due to lymphoid hyperplasia and boggy orifices and in treating sticking secretions in the tubes. It works more often with children than with adults. He believes that the main effect of the radiotherapy is the reduction of inflammation in the subepithelial tissue in the nose, with consequent lessening of secretion and no direct destruction of lymphoid tissue by the rays. Most benefit was obtained in cases in which there were minimal losses of not too long duration, with either a flat audiogram or an audiogram in which loss for the high notes was only 10 to 20 decibels less than that for the low octaves. A few good results were obtained in cases of profound loss for both air and bone conduction, but in cases of severe nerve deafness, especially those with a sharp drop or a dip in the curve of hearing, radiotherapy usually had no effect, even though there was much hypertrophy of lymphoid tissue about the eustachian tubes.

Emerson and associates [39] present a new applicator for irradiation of lymphoid tissue in and about the eustachian tubes in children for the treatment of deafness. With this applicator a high proportion of beta rays are in actual contact with the area to be treated. As a result, with 25 mg. of radium a twelve minute application delivers 5 milligram hours, which is sufficient to cause regression of the hyperplastic tissue. The authors describe the characteristics of this type of deafness, which Crowe and his co-workers have presented repeatedly. The insidious onset and the necessity for examination with the audiometer are stressed. One should eliminate preexisting infections of the upper respiratory tract and should perform tonsillectomy and adenoidectomy first when indicated. A nasopharyngoscopic examination is done with the use of pontocaine anesthesia, and the treatment is also carried out with topical anesthesia. Audiograms in 6 cases picked at random from a group of almost 100 cases showed definite and almost complete recovery of hearing. Even though the return of hearing may not be complete in some cases, arrest of the process may prevent serious deafness in adult life.

39. Emerson, E. B., Jr.; Dowdy, A. H., and Heatly, C. A.: Use of Radium in Treatment of Deafness by Irradiation, Arch. Otolaryng. **35**:845 (June) 1942.

Crowe and his co-workers [40] give a follow-up report of the results obtained with 152 children from irradiation of the nasopharynx according to the method described by Burnan. These children all had the typical loss for high tones due to lymphoid hyperplasia near the nasopharyngeal orifices of the eustachian tubes. They were followed over a two year period and have had from eight to twelve examinations each. One hundred and thirty-six of these children, or 89.5 per cent, now have normal tubal orifices, with the adenoids either small or absent. Only 16 had enough adenoid tissue left to require further treatment. The average number of treatments was slightly less than four, and only a few have had more than six treatments. The hearing acuity for high tones definitely improved in about one third of the ears having hearing defects limited to this range of frequencies. In slightly less than one third of the ears the amount of impairment remained the same. For the latter group the diagnosis remains uncertain, and the cause of impairment may be either a stationary lesion of the inner ear or an irreversible lesion of the middle ear. The advantage of treatment of this group may be the lessening of a possible combined type of deafness or the arrest of a potentially progressive lesion of the middle ear. Slightly over one third of the ears that had impaired hearing for some or all of the high tones at the first test have shown increase in the impairment during the two year period in spite of regression of adenoids and other nasopharyngeal lymphoid tissue. This type of deafness probably is due to progressive atrophy of the nerve or of the nerve and the organ of Corti in the basal turn of the cochlea.

A group of children who were not treated were also examined and carefully tested five times during the two years. Thirty-one per cent of this group had spontaneous regression of the lymphoid tissue, with both tubes clear, and an additional 15 per cent had one tube clear of lymphoid tissue. Their hearing at the onset was either good or only slightly to moderately impaired for the high tones. This group is not considered a control. There was slight improvement in response for the low frequencies of 32 to 256 cycles per second, the increase averaging from 1.5 to 4 decibels. For the middle range the average change was less than 1 decibel, and for the high frequencies of 4096 cycles per second and above the average impairment at the last test was greater than at the first test. For the boys the change averaged over 2 decibels at the first test, and for the girls, less than half that amount. At the first examinations defective hearing for high tones was found to be present more often in boys than in girls, and the impairment was present more often in children 11 to 13 years of age than in those 8 to 10 years

40. Crowe, S. J.; Guild, S. R.; Langer, E.; Loch, W. E., and Robbins, M. H.: Impaired Hearing in School Children, Laryngoscope **52**:790 (Oct.) 1942.

of age. The changes for the middle and lower ranges for the entire group of treated children having only loss for high tones were similar to those for the untreated group.

The group of children attending regular school classes who have poor hearing for tones other than the high ones, with thresholds down to 25 to 40 decibels, and somewhat higher for the high tones, due frequently to otitis media or scarred tympanic membranes with a history of otitis media, derive the most immediate benefit from radon therapy. Improvement in hearing of from 15 to 30 decibels occurs for all except the very highest tones. This produces good practical hearing from an educational standpoint. In the children in this group who did not improve failure was due to chronic suppurative otitis media or to extreme lesions of the middle ear resulting from previous repeated attacks of otitis, and no benefit was obtained even though the nasopharynx and the ears assumed an improved condition after radon therapy.

The authors feel that a large number of handicapping degrees of deafness could be prevented annually by prompt medical care in all new cases of acute otitis media. Proper education of the lay public, educators, school nurses, etc., would aid in accomplishing this. The authors conclude that radon therapy is a simple, safe and economical method for treating lymphoid hyperplasia of the nasopharynx and about the orifices of the eustachian tubes. Many children with impairment for conversational voice due to such lymphoid hyperplasia can have their hearing restored, and thus the educational problem is simplified for them, their parents and their communities. Only simple tests are necessary to discover the impairment of hearing in this group. For children with impairment of hearing for high tones only, careful examination with a good audiometer is necessary. Only under such circumstances in which careful work can be done and repeated examinations and proper follow-up observations can be made can a program of prophylactic treatment be carried out. The application of radon to the hyperplastic lymphoid tissue in the nasopharynx will be worth while in this group. Although the results are not spectacular, because the children have shown no definite impairment of hearing before therapy, the results are well worth the effort. The authors hope that this report will serve to encourage persons to undertake practical programs for the prevention of deafness among the children of their own communities. To succeed, each program must be carefully planned and adapted to the personnel and the facilities of that community.

Campbell [41] has made a critical and thorough analysis of the labyrinth fenestration operation. He reviews the early results of the operation

41. Campbell, E. H.: The Evaluation of the Labyrinth Fenestration Operation for Chronic Progressive Deafness, Laryngoscope **51**:1009 (Nov.) 1941.

and reports his own observations. To evaluate the results properly, sufficient time must elapse after the operation. Cases should be separated into two general groups, those with suitable and those with unsuitable indications for operation. The cases should also be separated into groups according to the operative technic. Changes in hearing should be calculated on the basis of average audiometric readings, particularly for the conversational frequencies 512, 1024 and 2048. The audiometric averages for the normal ear should also be taken into consideration. Psychologic factors should be evaluated, and the subjective reactions of the patient should also be considered. Of 30 cases in which the author performed operation, indications were suitable in 21. In 13 of these 21 cases improvement of more than 14 decibels was obtained, with an average improvement in hearing of 21.8 decibels. In the 8 cases in which operation was unsuccessful, an average loss of 4.8 decibels over the preoperative level resulted. In cases in which revision was done, the results were not successful. In 9 of the 13 cases in which the operation resulted in improvement, the intact ear showed a gain of 3.6 decibels over the preoperative level, and in 4 cases there was a gain of 3.5 decibels.

Campbell feels that there still exists much uncertainty regarding such features as indications for operation, accuracy of diagnosis, physiologic explanation of the results, desirability of particular technics and prognosis after operation. It is his personal opinion that the improvement obtained is of a practical degree in a sufficiently high percentage of cases to justify the procedure's being considered a practical measure. The end result should not be evaluated until an interval of four to eight months after operation. The author believes that the postauricular approach gives several important advantages over the endaural route. Further experimental and clinical research is necessary to clarify the technical and physiologic problems connected with the work.

Shambaugh [42] reports his results in 141 consecutive fenestration operations in which a modified technic, using constant irrigation and microscopic inspection, was employed. A significant improvement in hearing was obtained in 75 per cent of cases. A six month period should elapse after operation before a hearing result is regarded as "final" and probably permanent. In 87 of 103 cases in which revision or operation on both ears was done between July 1938 and September 1941 an average hearing gain of 24.4 decibels was obtained for the speech frequencies. Shambaugh has found that when hearing for the

42. Shambaugh, G. E., Jr.: A Modified Fenestration Technic: Analysis of Results in One Hundred and Forty-One Consecutive Operations, Arch. Otolaryng. **36**:23 (July) 1942.

speech frequencies is restored to within 20 decibels the patient will regard himself as having essentially normal hearing. When it is restored to within 30 decibels he will be able to meet the majority of social and economic situations without a serious handicap. He feels that patients with even a profound loss of 70, 80 or 90 decibels should not be refused operation because they cannot be given a hearing level of 20 to 30 decibels. A 30 decibel gain may mean as much to such profoundly deafened persons as to persons with a 40 or 50 decibel loss. It is necessary that the bone conduction be relatively good and that the patients understand that the most they can hope for is a small improvement in hearing. Persons with slight losses of hearing should not be operated on, as the operation will not give enough practical improvement to be worth the risk. There is always the possibility that the hearing may be made worse instead of better. For that reason one should never operate on the better hearing ear. Also, patients with primary nerve deafness or those with poor bone conduction should not be operated on.

Shambaugh [43] also reported his results with the fenestration operation before the Illinois State Medical Society. His series included a total of 146 operations on 116 patients. In 19 patients the original operation was revised because of closure of the fenestra. Eleven patients had both ears operated on. One hundred and three of the 116 patients, or 89 per cent, now have an improvement in hearing for conversational tones of more than 10 decibels above the preoperative level. The average gain is 26.5 decibels, and the greatest gain in any case is 45 decibels. In 9 patients the hearing is essentially unchanged. In 88 per cent of the cases the hearing has remained good for more than six months.

Priest [44] reviews a large series of cases of acute surgical mastoiditis at the University of Minnesota Hospital with special reference to postoperative hearing. In a series of 55 cases of acute surgical mastoiditis determinations of the hearing a few days before the operation and after operation were made. Unfortunately, the hearing in these cases was not and could not be determined before the otitis media began. The average hearing level curves in these cases showed a preoperative loss of 35 decibels for the lower frequencies, increasing to 45 decibels for the upper part of the scale. This improved after operation to a 25 decibel loss for the 64 and 128 frequencies, a 15 decibel loss for the middle range and a 35 decibel loss for the 4096 and 8192 frequencies. In a group of cases of acute exacerbation of chronic mastoiditis which was treated by complete simple mastoidectomy rather than by a radical operation, the hearing improved from 5 to 10 decibels for each frequency.

43. Shambaugh, G. E., Jr.: The Surgical Treatment of Deafness, Illinois M. J. **81**:104 (Feb.) 1942.

44. Priest, R. E.: Acute Surgical Mastoiditis, Arch. Otolaryng. **35**:250 (Feb.) 1942.

Priest concludes that in all types of deafness the hearing was improved after operation, with the greatest improvement in those cases in which the ears were operated on during a first attack of otitis media with surgical mastoiditis.

Shambaugh and Jennes [45] treated a series of 5 patients with progressive nerve deafness and tinnitus aurium with daily intravenous injections of 50 mg. of thiamine hydrochloride over a period of two weeks. No patient experienced any subjective or objective improvement in the hearing or in relief of the tinnitus. A review of the literature on the use of thiamine hydrochloride for the alleviation of deafness and tinnitus aurium shows no conclusive evidence in favor of the use of the substance for this purpose.

Schluederberg [46] reports his results with the use of prostigmine in the treatment of deafness and tinnitus aurium. Thirty-eight patients were treated, 20 for three months or longer, 16 for two months or longer and 2 for less than two months. The treatment consisted of semiweekly injections of 1 cc. of prostigmine methylsulfate (1:2,000) hypodermically and administration of 1 tablet (15 mg.) of prostigmine bromide orally three times daily after meals on the days when the injections were not given. Twenty of the 38 patients had perceptive deafness, 8 conductive deafness and 10 mixed deafness. Five, or 14 per cent, showed improvement in hearing. Of the 5 patients, 2 had the perceptive type, 1 the conductive type and 2 the mixed type of deafness. Ten, or 33 per cent, of the 30 patients who had tinnitus were improved, 2 of whom, with perceptive deafness, had complete cessation of this symptom. Of the other 8, 3 had the perceptive type, 2 the conductive type and 3 the mixed type of deafness. Five patients in this group had positive serologic reactions. One of these was completely relieved of the tinnitus, and another showed remarkable improvement in hearing and in the tinnitus. Improvement in hearing was considered only when there was a gain of 15 decibels or more on the average for the tonal range of 512, 1024 and 2048 double vibrations. Schleuderberg says that the improvement in hearing is due to the improvement in tinnitus. He also states that the conclusions drawn in this paper are at variance with those of a previous paper in which the same case reports were part of a larger group.

Judge [47] reports his results with the use of prostigmine in the treatment of chronic deafness and tinnitus aurium in a series of 20 patients.

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45. Shambaugh, G. E., Jr., and Jennes, M. L.: Therapy of Nerve Deafness and Tinnitus Aurium, Arch. Otolaryng. **35**:513 (April) 1942.

46. Schluederberg, H.: The Treatment of Deafness and Tinnitus Aurium with Prostigmin, Ann. Otol., Rhin. & Laryng. **51**:526 (June) 1942.

47. Judge, A. F.: Prostigmin in the Treatment of Chronic Deafness and Tinnitus Aurium, Mil. Surgeon **90**:177 (Feb.) 1942.

The duration of the disorder varied from eight months to two years or less. The plan of therapy was essentially the hypodermic injection of 1 cc. of a 1:2,000 solution of prostigmine methylsulfate three times a week and the oral administration of prostigmine bromide in doses of 15 mg. twice a day. The duration of the treatment is not mentioned. Local therapy, such as inflation and nasal treatments, was employed when indicated. Of the 14 patients having tinnitus, 64 per cent obtained complete or pronounced improvement, 21 per cent had some relief and 15 per cent failed to improve. Seventeen of the patients with impaired hearing showed significant audiographic improvement. The author concludes that prostigmine is of definite value in the treatment of tinnitus aurium. Although its value in treatment of deafness is questionable, its use should not be abandoned because of the paucity of other therapeutic agents available for the treatment of impaired hearing.

No comment is made on the diagnosis of the various hearing defects treated. It is difficult to evaluate results unless one has a thorough conception of the condition being treated. If the disturbance is a tubal or catarrhal involvement of the middle ear, one can understand and expect improvement from any therapeutic procedure, whereas impairment of perceptive hearing may not react favorably.

HEARING AND THE WAR

Simpson [48] emphasizes the importance of good hearing in modern service aviation because of the extensive use of various forms of telecommunication during flights. As a result aviators must be able to hear efficiently under the most difficult circumstances. The usual hearing tests as employed now are not entirely satisfactory. The forced whisper test is too variable because of the different environments under which the test is made and the different examiners who give the test. Pure tone audiometry is too lengthy for routine purposes and does not give any clue to the factor of quickness of interpretation in hearing. Changes in altitude produce the most common and most dramatic disturbances of the ear and nose. Simpson mentions a severe and permanent form of deafness as a complication of aero-otitis. After a few days the drum membrane appears normal, and the tubes are patent, but there is a high degree of deafness of the nerve or inner ear type, which may remain permanent. The vestibular reactions are not impaired.

Disease of the nose and throat will impair the efficiency of an aviator. Different views may be taken of the same disability according to whether it occurs in personnel undergoing training or in an experienced air crew. Actual suppuration of the ear in general is

48. Simpson, J. F.: A General Survey of Otorhinological Considerations in Service Aviation, J. Laryng. & Otol. **57**:1 (Jan.) 1942.

regarded as a contraindication to flying, if only from the point of view that the hearing is affected. Also, a dry perforation normally means deficient hearing and a chance for recurrence of otorrhea under the conditions of service. However, experienced air men so affected are used in nonoperational work. Moderate scarring of the drum membranes does not debar from flying provided the eustachian tubes are patent. A past history of suppuration of the middle ear should make one suspicious of some residual impairment in function of the eustachian tube. Simpson also mentions the various nasal and sinal symptoms and the complications occurring in flying. He states that it is remarkable that more aural and nasal disturbances do not occur in view of the number of men flying and the strain imposed on them. The rarity of such conditions may be due to the careful selection of the flying personnel.

Dickson [49] reports his observations on changes in hearing produced by aviation noise arising chiefly from three sources: the engine, the propeller and the wind. The intensity level of such noises is in the region of 120 to 130 phons. His observations coincide with those of other investigators in that the past evidence of such trauma manifests itself as a dip localized at 4096 cycles for both air and bone conduction. Repeated and continuous exposure to noise may involve frequencies below this level. No auditory defect may be noticed when the defect is localized at 4096 cycles, but hearing will be definitely impaired when the lower frequencies are involved. Mistakes in hearing the spoken word often occur when accurate interpretation depends on the ability to recognize consonants the essential characteristics of which are high frequency and weak intensity. A 50 to 60 decibel loss at 4096 cycles may produce difficulty in hearing such whispered words at 20 feet (6 meters) as "sister" or "solicitor."

The factors influencing the degree of hearing loss from acoustic trauma are: the total time of exposure; the length of exposure at each period; loud sounds; the character of the sound stimulus, whether continuous or interrupted; the type of protection adopted; the surroundings, whether enclosed or open (a smooth concrete wall will reflect 96 per cent of the acoustic energy striking it), and previous disease.

Analysis of airplane noise has shown that frequencies of the highest intensity, 110 to 115 decibels, are at the low end of the acoustic spectrum. This suggests that a low tone produces high tone deafness. Exposure of the ear to noise of pure tones has shown a maximum dip an octave below the fatiguing note. Also, sounds of low frequency produce less trauma than those of high frequency of approximately equal intensity.

49. Dickson, E. D. D.: Aviation Deafness and Its Prevention, J. Laryng. & Otol. **57**:8 (Jan.) 1942.

It is possible that the external auditory canal may act as a resonator, intensifying the frequency in a complex sound in the region of 3000 cycles per second, and thus cause a dip at 4096 cycles. It is also possible that sudden, instantaneous peaks of high intensity may occur at 2048 cycles at frequent intervals and thus be responsible for this dip.

The wearing of a flying helmet with telephones attached has been found to afford the fullest protection in the Royal Air Force. The characteristic dip after exposure to noise does not occur. Other forms of ear plugs, although of a protective nature, are usually impractical where the use of telecommunication becomes necessary. The wearing of the helmet with ear phones may damp out the high notes of high intensity, which are supposed to produce the greatest damage.

Fry [50] suggests a functional test of hearing for aviation candidates which will help select for flying duties men who will be worth training and to insure that no candidate is rejected who is capable of carrying out flying duties. Hearing in the flying service is important for the recognition and interpretation of auditory signals, whether Morse or speech. A person may have a gross hearing loss in one ear and still be as good in recognizing signals as a normal person with no hearing loss. The ability to interpret signals depends on both hearing and intelligence. This may be developed to a certain extent in training. The conditions under which the candidate has to hear, such as a noise background which may reach 120 phons, must also be considered. The test for pure tone thresholds does not take any of these things into consideration.

The test which is now being employed in the experimental stages is as follows: The subject is placed in a high level noise field with his ears protected by the standard service flying helmet. Lists of words and sentences are then conveyed to him through a pair of telephone receivers carried in his helmet. An alternative test is being developed in which both the background noise and the speech signals are delivered into the candidate's telephone. This can be accomplished by using gramophone records in which the test words or sentences are spoken against a noise background. This test may be further modified by varying the levels of speech and noise. The advantages of using gramophone records and of testing against a noise background are that conditions for testing can be reasonably standardized and that a number of candidates can be examined at the same time.

Many questions concerning the functional aspect of hearing remain to be answered. Fry mentions a few of these. How far can training compensate for defects of hearing, as shown by pure tone audiometry? How much more difficult is it to train a person with defective hearing

50. Fry, D. B.: A Suggestion for a New Method of Testing Hearing in Aviation Candidates, J. Laryng. & Otol. **57**:11 (Jan.) 1942.

than one with normal hearing? What is the effect of monaural deafness on the ability to receive signals? What is the effect of limitations of frequency and intensity in the transmission system on the reception of signals by a person with defective hearing? The answers to these questions are urgently needed.

Bunch [51] concludes that the loud noises of airplanes and airplane motors often cause hearing losses in pilots, especially for tones near 2048 and 4096 double vibrations. This impairment progresses with continued exposure, when the acuity for tones of lower pitch is also affected. Not all pilots are affected to the same degree. The flier who has decreased acuity for tones near 1024 double vibrations may have difficulty in understanding certain words over the radio and may not be able to understand exact landing instructions. Also, the pilot with hearing loss near 1024 double vibrations may have difficulty in following a radio guide beam if its frequency is near 1024 double vibrations and may have to keep his radio tuned on louder than is ordinarily required. If this is necessary, additional static, such as that created by lightning, may cause additional temporary or permanent hearing losses and incapacitate the pilot to such an extent that he may not be able to hear the radio beam. Bunch suggests that such possibilities may account for accidents which frequently occur during a storm. The pilot may not be aware of any hearing impairment, and the defect will often not be detectable with ordinary spoken voice tests. Frequent accurate audiometric tests by competent examiners should be made to solve this problem. A systematic study of the hearing of aviators would settle various disputes. It could determine whether certain fliers are more susceptible to the noise of airplanes than others, whether certain types of planes or motors are more harmful to hearing than others, whether it is possible to develop adequate protective devices for the ears, whether certain candidates should be excused from this type of training because they are more susceptible than others to the effects of noise and whether selection and protection cannot raise the efficiency and safety of the flying services. The hearing loss in aviators is not unlike that found in other persons constantly exposed to sounds of great intensity.

The audiogram of any flier, according to Campbell,[52] reflects the influence of six forces, which may not be associated with flying at all. These forces are the hearing apparatus which the flier inherited; his age; the effect of noise to which he has been subjected during his life; the ability of his tubotympanic apparatus to equalize changes in barometric pressure to which he has been subjected; disease, past and

51. Bunch, C. C.: Problem of Deafness in Aviators, War Med. **1**:873 (Nov.) 1941.

52. Campbell, P. A.: The Effect of Flight upon Hearing, Aviation Med. **13**:56 (March) 1942.

present, and the effects of anoxia. One may inherit a resistant or a susceptible acoustic mechanism, which may withstand or be easily affected by acoustic insults normally encountered in life. Age is a factor in normally depressing the upper tone limit. Noise and other vibratory energy will produce auditory fatigue and may result in changes in hearing. In flying, the energies producing this effect are engine explosions, propeller hums, slip stream effects on structures of the aircraft, sounds from moving parts, preaudible positive and negative pressure changes from motion of the propeller and suprasonic vibratory frequencies produced by objects moving at high speed and their overtones. The effect on hearing is the notching at the 4096 frequency, or, more correctly, the area from 4000 to 6000 double vibrations. This notch deepens and spreads fanwise until it involves the conversational range. Small insults produce transitory effects, and greater and prolonged insults result in greater changes, which may become permanent. Campbell has drawn charts showing the effects of noise, barometric changes and age on hearing.

Campbell [53] adds that experience has shown that a candidate can become a good air man if he simply possesses a nose of normal function, average normal ears and hearing and eustachian tubes and sinuses without obstruction. Hearing is essential for weather reports, interplane communication and detection of changes in motor rhythm. There is a constant alteration in the curve of hearing with changes in altitude and with fatigue of the organ of Corti produced by the 80 to 110 decibels or more of noise to which it is subjected. He explains the mechanics involved in the production of aero-otitis-media.

When the negative pressure inside the middle ear reaches 100 to 120 mm. of mercury the pain rather suddenly becomes severe. A loss of 10 or more decibels of hearing occurs usually in some portion of the low tone range of 128 to 2048 double vibrations. The drum membrane becomes sharply retracted, with engorgement of vessels along the handle of the malleus, followed by ecchymosis in Shrapnell's membrane and the lower aspect of the handle of the malleus. Serosanguineous fluid and bubbles form in the middle ear after one to eight or ten hours. Recovery may take place in from three to twenty-one days.

Campbell also describes a complication of external otitis from various forms of mycosis. Bullous myringitis followed by a collection of pus between the layers of the tympanic membrane results. Air sickness, aerosinusitis and the effects of altitude on the quality of the voice are discussed. No definite changes in the audiograms have as yet been noticed under conditions of anoxia produced in a pressure chamber at high altitude. Tests for spoken words instead of pure tones may show some changes and are to be made.

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53. Campbell, P. A.: Otolaryngological Problems of Aviation in World War II, Ann. Otol., Rhin. & Laryng. **51**:293 (June) 1942.

In his article on otolaryngologic disturbances occurring in fliers, Carson [54] presents observations which coincide with those of other investigators. He terms the impairment in hearing following prolonged flights fatigue of the auditory function. Time is a factor in such injury. Exposure to noise with an intensity of 110 decibels for twenty seconds may cause no demonstrable change in the organ of Corti in experimental animals; an intensity of 120 decibels for this time may cause microscopic evidence of damage, and an intensity of 120 decibels for thirty to forty seconds causes much more evident injury. Hearing losses shown by pilots after years of flying are principally in the range of audiometric frequencies of 1024 to 4096 or higher. The author feels that with cowling now provided for the cockpits and more flying being done in larger enclosed aircraft, damage to hearing should be less than it was a few years ago. He believes that headset noises and static are much more disturbing than any other noises. The head phone static frequently reaches the level of a sharply painful stimulus. He feels that loss of hearing acuity for higher frequencies is principally due to this one factor. If science can determine whether sounds of low pitch but high intensity are productive of greater fatigue than high-pitched sounds of great intensity, the problem might be attacked selectively by engineers and wear and tear on the tender subjectivity of man might be minimized. Progressive otosclerosis is neither benefited nor influenced in its inevitable course by flying. Carson also discusses problems affecting the vestibular system, the nose and accessory sinuses and the mouth and pharynx.

Fenton [55] compliments the flight surgeons for the excellent work they are doing and for the relatively simple and workable standards that they have established. He reviews the military physical standards required of pilots. Only those referring to the hearing will be summarized here. The necessity for patent eustachian tubes and nasal passages, free open external auditory canals and normal imperforate drum membranes is mentioned. Hearing must be 20/20 for the whispered voice for each ear on the original examination. Expert pilots on reexamination, observers and other nonpiloting personnel of the air force are permitted 8/20 for each ear, or 15/20 for one ear and 5/20 for the other. When audiometric examinations can be made, the average hearing loss permissible for pilots on original examination must not exceed 15 per cent. Good hearing is essential for the aviator because

54. Carson, L. D.: Otolaryngologic Problems Occuring in Fliers, J. A. M. A. **120**:4 (Sept. 5) 1942; Otolaryngological Aspects of Aviation, Laryngoscope **52**:704 (Sept.) 1942.

55. Fenton, R. A.: Otology and Aviation, Ann. Otol., Rhin. & Laryng. **51**:333 (June) 1942.

he depends on spoken or signaled orders by radio. Effects of flight on hearing depend on noise and vibration and on changes in air pressure while the plane is ascending or descending. The noise is somewhat masked by the pilot's helmet and rubber-cupped radio headset.

Older pilots show a sudden dip in the audiogram at about 4096 double vibrations, which may become permanent with repeated exposure to the fatigue produced by these noises. Changes in air pressure interfere with hearing for the lower range of conversational tones. During rapid descent, if the speed is greater than 300 feet (90 meters) per minute, the tubes are opened with increasing difficulty by swallowing, and severe pain, local congestion, transudation or hemorrhage may occur, with production of low tone deafness of various degrees. This condition is known as acute aero-otitis-media. Repeated exposure to this hazard may produce thickening of the lining of the tympanum and the eustachian tubes, with eventual formation of connective tissue and considerable reduction in hearing for low tones. Rapid negative acceleration, as in power diving, produces passive hyperemia of the eustachian tubes and tympanic structures, increases intracranial pressure and may conceivably produce minute hemorrhages in the end organs of hearing and equilibrium. Fenton also discusses the vestibular tests used and explains the various disturbances of equilibrium encountered in the flight. He states that otologists in general should acknowledge their debt to America's pioneer flight surgeons, notably Bauer, Tuttle, Armstrong, Campbell and Hargreaves, for their practical clarification of many of the problems of aviation, for passenger and pilot alike.

Sutherland [56] reports similar audiometric observations on 10 naval aviators who were tested before and after certain periods of flying. Most of the audiograms showed a severe loss of hearing for frequencies in the upper part of the scale, above 2000 cycles. With but 2 exceptions, the acuity for low tones was not considerably impaired. The author is now making further studies of improved helmets and screening devices for higher frequencies.

McGibbon [57] reports his observations on 100 men with aviation pressure deafness, known as aero-otitis. Ninety-four of the 100 patients complained of deafness of the conductive type, particularly for the frequencies of C and C_1 tuning forks, and audiometric readings showed a loss of 10 to 30 decibels for these frequencies. Although some subjects could still hear a whispered voice at 20 feet (6 meters), they showed impairment of hearing. An invagination of the tympanic membrane was the most common lesion observed. This was accompanied by con-

56. Sutherland, J. M.: Medical Research and War Problems, Ann. Otol., Rhin. & Laryng. **51**:445 (June) 1942.

57. McGibbon, J. E. G.: Aviation Pressure Deafness, J. Laryng. & Otol. **57**:14 (Jan.) 1942.

gestion of the region of the handle of the malleus and the vessels which sweep backward to the posterior malleolar fold. Other changes which followed were congestion of the attic, in 6 cases; congestion of the entire drum head, in 8 cases; effusion in the tympanum, in 6 cases, and rupture of the drum head, in 7 cases. The impairment of hearing was the last symptom to disappear. Nonventilation of the middle ear may be due either to failure or to inability to open the tube. McGibbon considers that tubal occlusion is brought on by two factors. One is the primary (pressure) occlusion, due to pressing together of the cartilaginous and the soft membranous walls. When this is neglected for several hours secondary (vital) occlusion occurs. This, he believes, may be due to the thickness of the submucosa and to the large numbers of mucous and goblet cells present in the epithelial lining of the cartilaginous tube. This secondary occlusion is a real entity and may take as long as fourteen weeks to resolve.

Poos [58] discusses the ear in aviation and warfare. He considers the effects of inadequate ventilation of the eustachian tubes, aero-otitis-media and the effect of noise on hearing. In a large series of aviators of different age groups the hearing loss was 5 to 10 per cent greater than that of a group of aviators of similar ages. With an increase of stratosphere flying and bomb diving a greater number of these defects in pilots may be expected. Poos mentions the effects of concussion and fracture of the skin on the internal ear. The practice of placing a pipe, pencil or comb between the teeth, or simply of opening the mouth and putting a finger in each ear when a bomb is expected to fall, has been found to diminish the incidence of injuries to the ear. A rubber mouth gag is being produced in Great Britain, as well as ear bandeaux of sponge rubber, ear plugs and other types of ear protectors to be worn as a safeguard against concussion during air bombardments.

Macfarlan [59] discusses the prevention and treatment of acoustic trauma. He suggests whenever possible the correction or abolition of the noises and concussions that cause such trauma. This, however, is impossible in most cases. The next step is such protection against noise as the wearing of ear plugs whenever possible and feasible. Those persons with labyrinthine nerve deafness, as well as those with chronic suppuration of the middle ear, should not be exposed to loud noises, either in civil or in military life. He also mentions the acoustic trauma resulting from the rapid rate of fire of some modern war weapons, which does not permit the stapedius muscle to act as a protector. The impairment of hearing in aviators is also mentioned, and the use of helmets

58. Poos, E. E.: The Ear in Aviation and Warfare, Eye, Ear, Nose & Throat Monthly **20**:342 (Dec.) 1941.

59. Macfarlan, D.: Prevention and Treatment of Acoustic Trauma in War and Civil Life, Laryngoscope **51**:964 (Oct.) 1941.

and rubber-capped head phones is recommended to reduce the outside noise. Efforts should be made to establish the more universal use of ear plugs for all persons when under fire and during air raids.

Shilling and Everley [60] have made a rather exhaustive study of the effect of various naval hazards on hearing, and they report their results in a series of articles. Their first paper deals with the effect of exposure to the noise of Diesel engines as experienced by men on submarine duty. Audiograms were made before and after exposure to these engines. The loss of hearing following one hour of exposure averaged 10.4 decibels for the entire group. The audiograms were similar to those made after other types of acoustic trauma, with the main dip in the curve occurring at 4096 cycles. The chief loss occurred after one hour of exposure, and only a comparatively slight increase followed the two, three and four hour exposures. The time of recovery, however, was greatly lengthened by increasing the time of exposure. Recovery was usually complete in five hours after one hour of exposure, whereas the hearing might still be impaired as long as twenty hours after two, three and four hour exposures. Fifty-eight machinist's mates who had been stationed on submarines were tested to determine permanent loss of hearing. The composite curve for this group showed loss of hearing for every frequency, but more evident at 2048, 4096 and 8192 cycles. This curve showed a remarkable resemblance to the audiograms of the men who had been given acute exposure to the noise of Diesel engines. The authors emphasize the importance of good hearing in any position in which vital orders have to be received, relayed or executed. They suggest that examination of the hearing should be given more weight in all physical examinations for selection of naval personnel. They recommend that quieting the Diesel engines in submarines as much as is feasibly possible would be worth while. Also, the use of ear plugs, such as the ear defenders, which produce a marked attenuation of noise, of about 30 decibels, without reducing the fidelity, would provide the necessary protection for the delicate mechanism of the ear against the trauma induced by the noise of the Diesel engines operating in confined space.

In the second part of their investigation, Shilling and Everley [61] report on hearing loss due to exposure to radio under service conditions. The hearing loss is significantly related to the duration of the exposure to radio work with ear phones. Men of ages from 15 to 24, with an average exposure to radio of thirty and three-tenths months showed an average hearing loss of 6.25 per cent; men of ages from 25 to 34, with an average exposure of eighty-five and three-tenths months, showed

60. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel, U. S. Nav. M. Bull. **40**:27 (Jan.) 1942.

61. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel, U. S. Nav. M. Bull. **40**:396 (April) 1942.

an average loss of 11.6 per cent, while men of ages from 35 to 44, with an average exposure of one hundred and forty-eight and two-tenths months, showed an average hearing loss of 14.4 per cent. The loss was apparent in the lower as well as in the higher frequencies throughout the entire range tested. However, in most instances the loss was considerably greater for the higher frequencies than for the lower ones, with the loss at 8192 cycles even greater than after exposure to the noise of Diesel engines. The loss of hearing is believed to be due to acoustic trauma produced by sudden exposure of the ear to loud sounds, such as that occurring when a nearby station comes in while the radio man is listening to a distant station. The power and sound may be so loud as to render the listener temporarily deaf, and several minutes may be required at times before recovery is sufficient to permit further routine work. Such trauma when repeated often may lead to permanent loss of hearing. The majority of ear phones issued to the naval service are particularly resonant at the midlow frequencies. This may account for the hearing loss for such frequencies observed in radio men, in addition to the impairment for higher frequencies.

Shilling and Everley emphasize the importance of constructing the radio equipment so as to eliminate the possibility of acoustic shock and trauma. They also learn from this investigation that proficient radio men need not have a naval audiogram. Excellent "sound" operators were found to have audiograms below normal in most cases. Ability to discriminate pitch is of primary importance for "sound" operators. Pitch discriminations are made with an audioscillator at three frequencies, 256, 1024 and 2048, which permits determinations to within 1 cycle above or below the reference notes mentioned. A study of this method as a supplementary examination for the selection of "sound" operators is now in progress.

In a third article,[62] Shilling and Everley investigated the effect of increased air pressure on the auditory mechanism. Examinations were made on candidates for "lung" training, who are subjected to 50 pounds (22.7 Kg.) of increased air pressure in the recompression chamber, required by training regulations prior to the candidate's entering the water for actual training with the submarine escape appliance. Fifty pounds of pressure is 2,585 mm. of mercury as compared with 380 mm. of mercury, or the difference between pressure at 18,000 feet (5.5 kilometers) and atmospheric pressure. In the group of 2,751 candidates who made this test, 152 cases of aero-otitis-media occurred. The complications varied from otitis media to mastoiditis, but the most serious was the associated loss of auditory acuity. There was an almost uniform

62. Shilling, C. W., and Everley, I. A.: Auditory Acuity in Submarine Personnel, U. S. Nav. M. Bull. **40**:664 (July) 1942.

loss of 20 decibels for each frequency greater than the average normal. The loss was of the conduction type. The degree of damage observed on otoscopic examination did not consistently reflect the loss of hearing shown by audiometric tests. The loss may at times be severe enough to be disabling and may take as long as a month to clear in favorable cases, and in some cases hearing may never return to normal. To determine the permanent effect on hearing due to exposure to increased air pressure, the hearing of divers was studied. These investigations showed little change in the hearing due to exposure to compressed air. The authors concluded that any changes in the hearing found in these divers is due to too rapid exposure to changes in pressure, producing improper tubal ventilation, etc. Exposure to compressed air in itself does not adversely affect the hearing. The authors stress the importance of great care in the physical examination and of proper instruction of the men prior to the "taking of pressure" in regard to swallowing, yawning and the use of the Valsalva inflation. The employment of helium and oxygen in aiding men to take the pressure test is also advocated. When extensive exposure to pressure is anticipated, a preexposure audiogram should be made as a safeguard to both workman and employer. Shilling and Everley are continuing their auditory investigations and will report further results in the near future.

Hartman [63] performed 35 experiments on hearing for upper tones by bone conduction on subjects in a pressure chamber. Under conditions of oxygen deprivation he found progressive lowering of the hearing for high tones as an expression of lowering of the general sensory functions. The hearing was restored by inhalation of oxygen.

Fowler [64] reviews the present hearing standards for the military services. In the Navy hearing of 15/15 is required for whispered voice in each ear, and binaural hearing must not fall below 7/15 for conversational voice during general service or below 7/15 for whispered voice in the aviation service. In the regular Army the standard is 20/20 for each ear. In the National Guard it is 20/20 in one ear and not less than 15/20 in the other. For flying cadets, class 1, hearing must be 20/20 in each ear, and for those of class 2 and 3, at least 8/20 in each ear. For class 1 the average hearing loss determined by audiometer is not more than 15 per cent, which is obtained by multiplying the average reading for frequencies of 512, 1024 and 2048 by 0.8. For selective service candidates, the low conversational voice is employed, and in case of doubt a watch is used. Class 1 A must have (*a*) normal

63. Hartman, H.: The Upper Tone Limit in Anoxia, Luftfahrtmedizin **3**:192 (Dec.) 1936.

64. Fowler, E. P.: Hearing Standards for Acceptance Disability Rating and Discharge in the Military Services and in Industry, Laryngoscope **51**:937 (Oct.) 1941.

hearing or (*b*) 10/20 or better in each ear. Class 1 B may have less than 10/20 but at least 5/20 in one ear. The other ear may be totally deaf. The watch and acumeter are sometimes used for testing. However, the voice is regarded as the most reliable test.

Fowler feels that some of the tests used are undependable and that for estimating percentage of disability within or without the services or in industry they are useless. Because of these tests there has been considerable dissatisfaction and controversy as to the amount of compensation due to veterans from loss of hearing in the first World War. The percentage loss as figured from the audiograms is not really representative of the actual loss of hearing because it does not consider the loss for actual speech. Some standard for measuring loss of capacity for hearing speech should be set up based on the way the ear hears speech and not on the way some one thinks it ought to hear speech. This not only would be invaluable for selecting candidates for military service but is well adapted for determining compensation of veterans for loss of hearing acquired during their military service. Fowler presents a chart, with his recommendations for suggested hearing standards for the military services. He also shows that the audiometer and the audiogram can be used as a true criterion for speech if proper interpretation, as well as proper weighting of the various pitches, is employed. Several items are essential in the construction of a percentage standard for hearing speech, both in civil and in military life. He emphasizes the use of an audiometer in a soundproof room, weighting of the various frequencies according to their importance for hearing speech and weighting for nerve deafness by the use of bone conduction as well as by air conduction measurements in order to make adjustments for the recruitment phenomenon. The construction of a table which weights the percentage of loss according to the degree of deafness is indicated. The table should be so constructed that even a layman can figure the percentage loss of capacity for monaural and binaural deafness. Fowler has constructed such a table and presents it with his article.

Mashburn and Marshall [65] compare the standards required of the British Royal Air Force and the United States Army Air Corps. Both services require that forced whisper of words, phrases or numbers be heard at 20 feet (6 meters). The patency of the eustachian tubes is tested by the Valsalvas maneuver or by an auscultation tube. Audiometer readings are not made in the Royal Air Force. Tuning forks, such as C (64 double vibrations), c_1 (256 double vibrations) and c_5 (4096 double vibrations), are used. The Rinne, Weber and

65. Mashburn, N. C., and Marshall, F. A.: Aviation Medical Standards, British R. A. F. vs. U. S. Army Air Corps, J. Aviation Med. **13**:62 (March) 1942.

Schwabach tests are made if required. The common otologic causes of rejection for flying are earache or discharge from the ears, deafness or noises in the ears and frequent colds.

Fox [66] discusses the tests he uses for determining defects in the ear, nose and throat in relation to recruitment in the Navy. For testing simulated deafness, repeated spoken and whispered voice tests are used with the subject's eyes covered and are repeated on different days if necessary, the distance at which the voice is heard at each test being noted. A quiet room, Fox states, is not necessary, but it should be free from excessive loud noises. A person who is simulating will hesitate in answering and will sometimes claim to hear only one of two numbers even if they are alike. In the loud voice test with the subject holding the finger in the good ear, if the subject refuses to repeat to a loud-spoken number when it is shouted in the good ear, he is malingering. The Lombard test with a Bárány noise apparatus is suggested in cases of absolute deafness in one ear. The Gault-Cochleo palpebral test for absolute simulated deafness in one or both ears can also be employed. If the noise used produces contraction of the eyelids the patient has hearing in that ear. The Rinne and Weber tests will also aid in testing malingering.

The tests mentioned by the author are not only elementary but far from being accurate. For superficial and quick testing for malingering these methods might be considered, but surely not for any accurate or scientific investigation.

In his past experience during the first World War, Lewis [67] found that hearing tests calculated to meet the demands of military service in many instances served to reject men well qualified for service and in other instances to pass men who were ill qualified. The otologic requirements differ widely for the various arms of the military service. Hearing which may be adequate for men in one branch may not be sufficient for those in another, such as a pilot or the soldier stationed at some listening post. In addition, the hearing may change after exposure to various noises accompanying warfare. Otology can only confirm the momentary adequacy of cochlear and vestibular function at any stage of an individual soldier's service. Quantitative or qualitative observations on cochlear and vestibular impairment can be considered permanent only when confirmed by repeated observations over sufficient intervals. In evaluating audiograms one must be aware of the discrepancies found in different audiometers. A loss of 10

66. Fox, F. G.: Eye, Ear, Nose and Throat Defects in Relation to Recruiting, Dis. Eye, Ear, Nose & Throat **2**:176 (June) 1942.

67. Lewis, E. R.: Malingering and Neuro-Otologic Considerations in Combat Services, Laryngoscope **51**:970 (Oct.) 1941.

decibels in the 1024 to 2048 region is not disqualifying. A 10 to 15 decibel loss for tones between 256 and 3000 double vibrations is not disqualifying for general military service. In addition, a loss of 25 decibels in the 128 double vibration region and as much as 35 to 40 decibels in regions below 96 double vibrations is also not disqualifying. Likewise, a hearing loss of as much as 25 decibels at 6,000 double vibrations and a 35 or 40 decibel loss for the higher frequencies may not be disqualifying except for special listening device services.

For precise measurement of hearing the audiometer under suitable conditions is the instrument of choice. It is impracticable under conditions of combat service. The only procedures required in war zones which can be carried on anywhere are the voice, whisper, tuning fork, watch tick, noise apparatus and caloric douche tests. Precision must yield to practicability under combat conditions. Malingering assumes varying aspects in military service, depending on circumstances. Lewis does not consider methods but stresses the importance of medical officers realizing fully the nature of their responsibility in establishing a diagnosis of malingering.

In a survey of 19,223 medical records of American men between the ages of 21 and 36 examined under the draft act, Rowntree and associates [68] report their results in order to provide an index to the physical fitness for military service of men of such ages. The sample was drawn from each state in proportion to total registration and is a cross section of registrants examined prior to May 31, 1941. Approximately 50 per cent of the 2,000,000 registrants were found to be unqualified for general military service, physically, mentally and educationally. Only about 100,000, however, were classified as unfit because of lack of educational requirements. Of the remaining 900,000, 470,000 were qualified for limited military service only, and 430,000 were totally disqualified for any military service. The estimated total who were unqualified for service because of defects of the ears was 41,000, or 4.6 per cent.

25 East Washington Street.

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68. Rowntree, L. G.; McGill, K. H., and Folk, O. H.: Health of Selective Service Registrants, J. A. M. A. **118**:1223 (April 4) 1942.

Abstracts from Current Literature

Ear

OSTEOMYELITIS OTHER THAN OF THE TEMPORAL BONE SECONDARILY AFFECTING THE COCHLEAR AND VESTIBULAR STRUCTURES: REPORT OF THREE CASES. MATTHEW S. ERSNER and JULIUS WINSTON, J. A. M. A. **117**:1619 (Nov. 8) 1941.

The authors discuss the occurrence of osteomyelitis of the temporal bone, in which the otic capsule and the vestibular and cochlear portions of the auditory nerve may become directly involved. They comment on the fact that in general osteomyelitis involving other structures than the temporal bone, the otic capsule and the auditory nerve may become affected secondarily by embolic phenomena or by contiguity or toxic absorption, with the creation of the symptoms of tinnitus, vertigo and deafness.

Several case reports are cited, together with audiometer charts, and certain important points are emphasized. The authors conclude as follows:

"1. Osteomyelitic deafness is probably not as rare as one has been led to believe. We believe that if hearing tests and otologic examinations were performed as a routine in every case of osteomyelitis the incidence of osteomyelitic deafness would be found to be more frequent than has been heretofore recognized.

"2. Postmortem studies of the temporal bone would be of value.

"3. Bezold's observation may lead to a clue in its relationship to other forms of deafness."

GORDON, Philadelphia.

THE SIGNIFICANCE OF AUDIBLE ONSET AS A CUE FOR SOUND LOCALIZATION. DAVID P. BODER and IRVING L. GOLDMAN, J. Exper. Psychol. **30**:262 (March) 1942.

The authors have made a series of experiments pertaining to sound localization (1) to determine the localization value of clear onset of tones versus the masked onset of tones of the same duration, (2) to compare the localization value of prolonged tones of either clear or masked onset with the localization value of an interrupted tone and (3) to compare responses under indoor and outdoor conditions.

From their experiments, they made the following conclusions: 1. Indoors, an intermittent tone of 680 double vibrations is localized better than a continuous tone of the same frequency with a masked onset, but there is only a tendency to better localization when compared with a continuous tone with a discrete onset. 2. A definite pattern is thus set up, namely, intermittent, discrete, continuous, which prevails in 44.8 per cent of cases. However, this relationship is frequently reversed. 3. The response to the outdoors experiment closely followed the formula noted in the indoors experiments. 4. Finally, the clear off-on response of the cortex to intermittent or discrete tones represents an important bioelectrical component of higher integrative processes, such as auditory space perception.

PERSKY, Philadelphia.

RECONSTRUCTIVE OTOPLASTY. PAUL W. GREELEY, Surgery **10**:457 (Sept.) 1941.

The chief stumbling-block in reconstructing a new ear lies in the problem of furnishing an adequate support to the overlying skin. Prior to the following procedure, the best attempt was the use of costal cartilage or iliac bone, which was shaped to represent the missing cartilage. The use of cartilage from the ear of another person, usually the mother, produces far better results. To remove

the maternal cartilage, an incision is made along the posterior border of the helix and the skin dissected off anteriorly and posteriorly to free the cartilage, which is then removed and the skin closed. A dressing on a modeling compound stent holds the skin flaps together accurately. There is much less deformity of the maternal ear than might be expected.

The cartilage is then buried under the skin at the site of the future ear and the incision allowed to heal. On the second stage of the operation, three to ten weeks later, the incision is reopened, the cartilage turned forward and the bare area covered with a skin graft on a modeling compound mold. The end result is superior to the use of any other supporting framework employed in the past.

H. A. MILLER, Philadelphia.

HEREDITARY SYPHILIS OF THE EAR. E. ESCAT, Oto-rhino-laryng. internat. **25**:113 (Aug.) 1941.

In 1930 Escat reported to the Societé de Médecine of Toulouse, France, his observations in 104 cases of hereditary syphilis of the auditory apparatus and compared these cases with 93 cases of acquired syphilitic otopathy. The essay was never published. In the ten years since 1930 he has observed 36 more cases. In the first group there were 81 cases of impairment of hearing from labyrinthtitis or from meningoneuritis; in the remainder the diagnosis was gumma of the tympanum and mastoid. The Bordet-Wassermann reaction was positive in 23 of 44 cases; the triad of Hutchinson was present in only 10 per cent of the 104 cases. The insidiousness of this form of syphilis, the sole symptom often being deafness, and the difficulties of diagnosis are emphasized. Characteristic are perceptive deafness, accompanied by early deficiency of bone conduction with relatively good hearing for voice, lack of tinnitus, presence of Hennebert's syndrome, paralyses of the oculomotor and facial nerves (more frequently seen than in other forms of otopathy) and, frequently, negative reactions to the Wassermann test.

Extreme caution must be exercised in the use of arsphenamine preparations because of the danger to Corti's organ. It is preferable to inaugurate the treatment with mercury or bismuth salts.

Combining the two groups of patients one finds that 77 of them were females and 63 males; 40 patients were improved, 12 notably. The relatively poor results of therapy in congenital aural syphilis are to be attributed to delay of diagnosis rather than to inefficacy of the methods of treatment.

DENNIS, San Diego, Calif.

LABYRINTHINE MENINGITIS CURED BY LABYRINTHECTOMY AND CHEMOTHERAPY. JUAN LAYERA, JUAN MANUEL TATO and TOMAS ITURRIOZ, Rev. otorrinolaring. **1**:30 (Sept.) 1941.

Scarlet fever in an 8 year old child was complicated by fulminating otitis media and mastoiditis. Simple mastoidectomy was performed, and four days later labyrinthitis developed with marked nystagmus, chills, fever, nausea and vomiting. The nystagmus was toward the normal ear and of horizontal-rotary character. Two days later, facial paralysis occurred. The labyrinth failed to give any response to the caloric test, which indicated suppuration within it. Roentgen examinations revealed a suppurative focus in the petrous apex. In consequence of this, a radical mastoid operation was performed, and pus was evacuated from a cavity in the vicinity of the horizontal semicircular canal. This canal showed destruction of its bony wall and a moderate amount of granulation tissue overlying the area. To combat the infection, septazine (N^4-benzylsulfanilamide) was given. On continuance of the infection and the symptoms plus the onset of drowsiness—although there was no frank indication of the meningeal involvement—spinal puncture was performed. This showed 500 cells per cubic centimeter, predominately polymorphonuclear leukocytes. The smear revealed occasional pneumococci and streptococci, but the culture showed a pure growth of streptococci.

In consequence, labyrinthectomy was performed according to the Jensen technic. Chemotherapy was given in addition, intraspinally, subcutaneously and orally.

The child was also given a blood transfusion (150 cc.). Three days after the operation all evidences of septicemia had disappeared, and the subsequent convalescence was uneventful.

In commenting on this case the authors point out (1) the severity of the infection with the extensive destruction of the mastoid process; (2) the formation of a petrous abscess with destruction of the horizontal semicircular canal; (3) the presence of facial palsy; (4) the presence of meningitis with a pure culture of streptococci. They point out the importance of careful observation and of proper timing of each operation, the favorable results obtained by operation and the role of chemotherapy. The latter must be advanced intensively by every known route of administration and the concentration of the drug in the blood and the spinal fluid should be maintained for a period sufficient not only to combat the original infection but to prevent any recurrence from the residual foci.

PERSKY, Philadelphia.

MASTOIDITIS AND THE SEDIMENTATION TEST. JUAN B. ARCIONI and HORACIA E. L. QUEROL, Rev. otorrinolaring. **1**:78 (Sept.) 1941.

Arcioni and Querol found that the sedimentation rate is always increased in surgical mastoiditis, running between 65 and 90 mm. and between 99 and 125 mm. for the first and second hours, respectively, with an average of 77.5 and 107 mm., respectively. This is true with respect to what they term the fibroconnective phlegmons. With empyema of the mastoid, the values range from 17 to 86 mm. and from 31 to 110 mm. for the first and second hours, respectively, with a mean of 51.5 and 70.5 mm. In the cases of thrombophlebitis they found the rate to be 35 mm. the first hour and from 43 to 60 mm. the second hour. Normally, fifteen to twenty days after the operation the sedimentation rate returns to normal. With any complication there is always an increase in the curve. They consider the normal rate as 10 mm. for the first hour and 14 mm. for the second hour. The clinical gravity, however, does not depend on the intensity of the drop in the sedimentation rate. The well pneumatized mastoid always gives a higher rate, while the sclerotic mastoid gives a normal and localized empyema usually gives a lower than normal rate.

PERSKY, Philadelphia.

Pharynx

ACUTE ABSCESS OF THE THROAT. WILLIAM DEERING and JOSEPH BRENNEMANN, J. A. M. A. **118**:1171 (April 4) 1942.

The authors report the observations on and conclusions derived from the study of a series of 250 cases of true and potential abscess of the throat in children under 14 years of age. They emphasize the fact that this study was conducted from the pediatric point of view because the service of the hospital is entirely devoted to pediatrics and all the residents of the house staff except the pathologist are in training for the practice of pediatrics. No resident in otolaryngology was on the staff during the period of the publication of this report or during the period of study, which is given as ten years.

This presentation is therefore given "in a simple clinical way, without anatomic or pathogenic detail, in the hope that [the observations] may be of interest and service to the pediatrician and general practitioner, and possibly to the laryngologist because of the method of approach."

Retropharyngeal, retrotonsillar, peritonsillar and parapharyngeal abscesses are discussed in moderate detail. In only 11 instances was an abscess found in a child on whom tonsillectomy and adenoidectomy had been performed; in 7 of these the abscess was retrotonsillar, in 2 peritonsillar and in 2 "uncertain."

Treatment was undertaken along tested procedures, and incision was performed when advanced fluctuation or pointing took place.

Every means in the pediatric armamentarium was thoroughly carried out, and all supportive measures were utilized. On a few occasions it was necessary to call in an otolaryngologist to incise an abscess.

Sulfanilamide was just beginning to be used at the time this study was made and was the only drug of its group employed. In the light of the limited experience with this substance, the authors venture the opinion that when suppuration has not as yet taken place sulfanilamide or one of its derivatives should be given in accurate dosage and a proper level of concentration maintained in the blood. On several occasions it was found that suppuration was temporarily delayed by the use of sulfanilamide and on withdrawal of this drug the abscess proceeded to pointing.

They summarize their findings as follows: "In a clinical study of 250 cases of true and potential, or abortive, abscess in children under 14 years of age there were 181 abscesses and 69 cases in which suppuration did not occur. There were 82 retropharyngeal, 79 retrotonsillar, 11 peritonsillar, 1 parapharyngeal abscess and 8 of undetermined classification. There were two deaths following retropharyngeal and one due to parapharyngeal abscess. The total mortality was 1.7 per cent; that following retropharyngeal abscess alone was 2.4 per cent. Only one death was unquestionably due to an abscess itself, the parapharyngeal abscess. Complicating cervical abscess occurred in 6.6 per cent of the series."

GORDON, Philadelphia.

Larynx

PARALYSIS OF THE LARYNX. LOUIS H. CLERF and OLIVER W. SUEHS, Ann. Otol., Rhin. & Laryng. **50**:762 (Sept.) 1941.

Laryngeal paralysis is most often due to a peripheral lesion, and only in 10 per cent of cases to a central lesion. All the laryngeal muscles except the cricothyroid are supplied by the recurrent laryngeal nerve; the arytaenoideus transversus, which is the only unpaired muscle, receives fibers from both sides. The cricothyroid muscle is supplied by the superior laryngeal nerve. The authors conclude, on the basis of their personal observations and the reports of other investigators, that injury to the recurrent laryngeal nerve results in partial adduction, with loss of tension of the corresponding vocal cord. It is possible that this is preceded by a brief period of full adduction. If recovery of function does not occur, the paralyzed cord assumes a median position, tension improves and the voice returns to normal. A vocal cord in the midline does not later assume the cadaveric position. The true cadaveric position is observed infrequently in cases of paralysis of the recurrent laryngeal nerve associated with loss of function of the corresponding superior laryngeal nerve. The occasional cadaveric position of the cord observed in cases of uncomplicated paralysis of the recurrent laryngeal nerve probably results from atrophy of the cricothyroid muscle.

M. V. MILLER, Philadelphia.

ULCERATIVE LARYNGITIS AS A COMPLICATION OF MEASLES. CARLOS PEDRO MERCADINO and DONATO VIVOLI, Rev. otorinolaring. **1**:38 (Sept.) 1941.

The authors discuss the pathology of ulcerative laryngitis occurring as a complication of measles. They consider, first, the appearance of the larynx in the stage of invasion. Here they observed merely a congestive phenomenon which extended into the larynx from the surrounding mucous membrane. The symptoms vary from mild hoarseness to severe stridulous laryngitis associated with dyspnea and severe fighting for breath. Second, during the stage of the eruption, the eruption may cover the entire larynx and become a grave complication, since it may produce infiltration about the aryepiglottic folds and even extend to the subglottic region. Here, too, dyspnea and asphyxia may be so severe that the differential diagnosis from diphtheria is often difficult. Third, during convalescence the lesion is usually that of ulcerative laryngitis. This as a rule appears at the conclusion of the period of eruption, and the symptoms are merely an aggravation

of those seen in the earlier stages. Hoarseness is present, progressing to complete aphonia; the cough is dry and painful; the expectoration is mucopurulent and often streaked with blood. The dyspnea, with pronounced supraclavicular retraction and rapid progression to suffocation, is evidence of severe inflammation of both the mucosa and the submucosa.

Pathologically, intense redness and swelling of the mucosa of the epiglottis, the false cords and the infraglottic region are noted. Small ulcers are often present on the posterior wall of the larynx. The ulcerations may become more or less extensive; they are always serpiginous and are usually localized to the posterior segments of the vocal cords and to the arytenoid cartilages. Simple erosions may be encountered on the free borders of the vocal cords, but if they are deep they may form small cavities in the vicinity of the arytenoid cartilages and the posterior plates of the cricoid cartilages and advance to the trachea.

The authors' observations are based on postmortem examinations in 5 cases of death, occurring in a series of 4,219 patients who were admitted to the Muñiz Hospital from 1927 to 1940. As a result of this study, the authors came to the following conclusions: 1. The laryngeal lesions precede the appearance of bronchopneumonia and are the direct cause of the bronchopneumonia. The pneumonia does not occur as a result either of an infection or of aspiration. 2. It is impossible to establish an etiologic classification of all the infections and eruptive diseases which can produce lesions of the larynx. This is probably due to the extreme polymorphic character of the lesions, which may range from simple congestion to severe ulceration. 3. Contrary to former opinion, the ulcerative lesions most frequently occur in the upper portion of the laryngotracheal tract. 4. It is difficult in some cases to make a differential diagnosis from diphtheritic croup. Direct smears and direct laryngoscopic examination may be necessary to establish the diagnosis. Lastly, in those cases in which there is severe ulceration of the larynx with extensive involvement of the trachea and associated bronchopneumonia, little can be accomplished by intubation, tracheotomy or oxygen therapy. In such cases the outcome is, as a rule, fatal.

PERSKY, Philadelphia.

Nose

MANAGEMENT OF THE NOSE AND PARANASAL SINUSES IN ASTHMA. AUSTIN SMITH, Pennsylvania M. J. **44**:41 (Oct.) 1941.

The author makes a plea for treatment by the allergist and the internist before local measures are instituted in the management of nasal and paranasal conditions associated with asthma. Allergic patients are divided into two groups: (1) the preasthmatic and (2) the asthmatic.

In group 1 are those patients who frequently come for "treatment of symptoms referable to the upper part of the respiratory tract, such as paroxysmal sneezing, blocked nose and rhinorrhea."

In group 2 are those "with established bronchial asthma and associated pathology in the nose and sinuses."

Allergic study must be thorough to be of real value. The author states that "the rhinologist and the practitioner have neither the time, the training, nor the expensive equipment necessary to make complete allergic studies or to carry out appropriate treatment." Cooperation between the rhinologist and the allergist is greatly desired. This is particularly applicable to children. Operations should be as conservative as possible and should be undertaken only when the allergy is under control.

WEINBURGER, Sayre, Pa.

BONE AND CARTILAGE TRANSPLANTS: THEIR USE AND BEHAVIOUR. RAINSFORD MOWLEM, Brit. J. Surg. **29**:182 (Oct.) 1941.

In nasal plastic surgery the use of cartilage as a transplant is vitiated by the tendency of approximately 25 per cent of these grafts to become distorted. So far no method of controlling this fault has been evolved.

Iliac bone survives transplantation most successfully. Roentgenographic and histologic follow-up leads to the belief that this type of graft does well regardless of whether the periosteum is saved or not and that it need not necessarily be in contact with other bone. In 102 cases there was union to the nasal bones in 66. There was no clinical or roentgenographic difference between those grafts which were adherent and those which were free. Aside from the slight alteration in the appearance of the transplant during the first two to three months, while irregularities tend to become rounded off, the graft persists indefinitely as a living graft in an unchanged state.

HENRY MILLER, Philadelphia.

CACOSMIA. A. ROSENZVIT, Rev. otorrinolaring. **1**:98 (Sept.) 1941.

Rosenzvit discusses the causes of cacosmia from both the objective and the subjective standpoint. Among objective causes he mentions foreign body in the nasal fossa; syphilitic coryza; sequestrum in the nasal fossa; maxillary sinusitis, especially of dental origin; caseous sinusitis and rhinitis; fetid secretions in the fossa of Rosenmüller and the pharyngeal bursa of Thornwaldt; ozena; lesions of the tonsils; dental caries; lesions of the tracheal and bronchopulmonary tracts; lesions of the digestive apparatus, and foreign bodies and calculi in the mouth or in the maxillary sinus. Subjective causes are sensory hallucinations of neurotic and psychopathic persons, auras of epileptic, neurasthenic and hypochondriac persons, tabes and dementia paralytica, hysteria and mental alienations, lesions of the olfactory nerve due to either grip or diphtheria, psychic conditions arising during pregnancy and sinusitis of nasal origin.

The author's contribution deals primarily with the presence of calculi in the maxillary sinus and also in the nasal fossa. He describes 2 cases in which calculi were diagnosed by roentgenogram and all symptoms were cleared up on removal of these foreign bodies.

PERSKY, Philadelphia.

Miscellaneous

EXPERIMENTAL STUDIES ON HEADACHE: FURTHER ANALYSIS OF HISTAMINE HEADACHE AND ITS PAIN PATHWAYS. G. A. SCHUMACHER, B. S. RAY and H. G. WOLFF, Arch. Neurol. & Psychiat. **44**:701 (Oct.) 1940.

The article is summarized by the authors as follows:

1. Of 15 subjects in whom afferent impulses from the superficial tissues of the scalp on one side were blocked by procaine, histamine headache in 4 and headache due to pneumoencephalographic examination in 11 were the same on the two sides of the head.

2. In 12 subjects obliteration of the circulation of the scalp by a blood pressure cuff modified the intensity of the histamine headache. That the reduction in pain was due to raising the pain threshold by introducing a second pain (the tight cuff) is offered as a likely hypothesis. Of 5 subjects, obliteration of the temporal artery by manual compression reduced the intensity of the histamine headache on that side in 2 but had no effect in 3. Complete subcutaneous infiltration with procaine hydrochloride of half the anterior part of the scalp (frontal, parietal and temporal areas on one side), including presumably all the vessels of the scalp in that area, did not prevent severe histamine headache in the frontotemporal region, bilaterally, which was of equal intensity on the two sides.

3. Injection of histamine directly into the temporal artery resulted in homolateral temporoparietal headache in 2 subjects.

4. Ligation of the temporal artery on one side in 4 subjects and of the middle meningeal artery on one side in 7 subjects did not influence the intensity of the headache experienced on the two sides after injection of histamine.

5. In 7 subjects who had partial section of the sensory root of the fifth cranial nerve on one side (resulting in unilateral analgesia of the lower part of the face in 6), histamine headache occurred bilaterally.

6. In 7 of 8 patients who had complete section of the sensory root of the fifth cranial nerve (resulting in unilateral hemianalgesia of the face and anterior half of the scalp), histamine headache was not induced on the denervated area, but did occur elsewhere in the head.

7. In a subject with complete hemianalgesia of the face and head (resulting from a head injury), strictly unilateral hemicrania on the normal side of the head was induced by histamine.

8. Histamine headache was absent in the back of the head on one side, but was present elsewhere in the head in 2 subjects with lesions of the cervical portion of the cord, causing unilateral occipital analgesia.

9. Section of the seventh and the ninth cranial nerve, respectively, in 2 subjects did not affect the bilateral equality of the headache induced by histamine.

10. Section of both sympathetic trunks in 2 subjects (cervical portion in 1 and thoracic portion in the other) had no effect on subsequently induced histamine headache.

The conclusions reached were as follows:

1. Histamine headache does not depend on the integrity of sensation from the superficial tissues.

2. The extracranial and dural arteries play a minor role in contributing to the pain of histamine headache.

3. Cerebral arteries, principally the large arteries at the base of the brain, including the internal carotid, the vertebral and the basilar artery and the proximal segments of their main branches, are chiefly responsible for the quality and intensity of histamine headache.

4. Although there may be other less important afferent pathways for the conduction of impulses interpreted as headache following injection of histamine, (*a*) the fifth cranial nerve on each side is the principal afferent pathway for headache resulting from dilatation of the supratentorial cerebral arteries and felt in the frontotemporoparietal region of the head, and (*b*) the upper cervical nerves are the most important afferent pathways for headache resulting from dilatation of arteries of the posterior fossa and felt in the occipital region of the head.

R. Irvine [Arch. Ophth.].

Experimental Studies on Headache: Further Analysis of the Mechanism of Headache in Migraine, Hypertension and Fever. A. M. Sutherland and H. G. Wolff, Arch. Neurol. & Psychiat. **44**:929 (Nov.) 1940.

Previously the authors have indicated that dilatation and distention of cranial arteries form the basis of headache produced by a variety of factors. The purpose of this communication is to analyze further the mechanism of such headache and to ascertain which cranial arteries are responsible in each instance.

For example, faradic stimulation of the proximal few centimeters of the anterior middle and posterior cerebral arteries and of the first few centimeters of the intercranial portion of the internal carotid artery causes pain within, behind or over the homolateral eye. Furthermore, stimulation of the vertebral and basilar arteries and the proximal portions of the branches causes pain in the occipital and the suboccipital region. These areas are commonly involved in attacks of migraine.

To correlate the data on the various headaches under consideration, a comparison of the prototypes of each class of headache was made. Histamine headache was taken as the representative of a class which includes the headaches induced by amyl nitrite and foreign proteins and those associated with fever. The migraine

headache was taken as representative of a class which includes, as well as the many varieties of migraine, the headache associated with hypertension.

After analyzing these various types of headache from the point of view of production, natural and experimental, and of alleviation, as by vasoconstrictor drugs and manual compression of vessels, they draw the following conclusions. For the histamine headache it is likely that the cerebral branches of the internal carotid, basilar and vertebral arteries at the base of the brain are primarily responsible. To the migraine headache, however, the extracranial, and possibly the dural, branches of the external carotid artery are the chief contributors.

R. IRVINE [ARCH. OPHTH.].

EXPERIMENTAL STUDIES ON HEADACHE: PAIN-SENSITIVE STRUCTURES OF THE HEAD AND THEIR SIGNIFICANCE IN HEADACHE. BRONSON S. RAY and HAROLD G. WOLFF, Arch. Surg. **41**:813 (Oct.) 1940.

In the course of a comprehensive inquiry into the cause of headache, Ray and Wolff tested out the sensitivity of the extracranial and intracranial structures to pain. A variety of stimuli were used. All the tissues covering the cranium, especially the arteries, are sensitive to pain. The cranium, including the diploic and emissary veins, the parenchyma of the brain, most of the dura, most of the pia-arachnoid, the ependymal lining of the ventricles and the choroid plexuses, is not sensitive to pain. The great venous sinuses and their venous tributaries from the surface of the brain, parts of the dura at the base, the dural arteries and the cerebral arteries at the base of the brain are sensitive to pain. Stimulation of the pain-sensitive structures on or above the superior surface of the tentorium cerebelli resulted in pain in various regions in front of a line drawn vertically from the ears across the top of the head. The pathways for this pain are contained in the fifth cranial nerve. Stimulation of the pain-sensitive intracranial structures on or below the inferior surface of the tentorium cerebelli resulted in pain in various regions behind the line just described. The pathways for this pain are contained chiefly in the ninth and tenth cranial nerves and the upper three cervical nerves.

From the data available, six basic mechanisms of headache have been formulated. Headache may result from (1) traction on the veins that pass to the venous sinuses from the surface of the brain and from displacement of the great venous sinuses, (2) traction on the middle meningeal arteries, (3) traction on the large arteries at the base of the brain and their main branches, (4) distention and dilatation of intracranial and extracranial arteries, (5) inflammation in or about any of the pain-sensitive structures of the head and (6) direct pressure by tumors on the cranial and cervical nerves containing many pain-afferent fibers from the head. Intracranial diseases commonly cause headache through more than one of these mechanisms and by involvement of more than one pain-sensitive structure. Traction, displacement, distention and inflammation of cranial vascular structures are chiefly responsible for headache. Headache from intracranial disease is usually referred pain. Local tenderness of the scalp may serve as an index to the structures responsible when a lesion produces direct irritation of pain-sensitive structures. Since disease of remotely separated pain-sensitive structures may cause pain and hyperalgesia in identical areas, the clinical usefulness of such localization is limited. But unilateral hyperalgesia localized in the parietal area indicates the possibility of a lesion near the middle meningeal artery, and hyperalgesia localized to the postauricular region indicates the possibility of a lesion in the region of the internal auditory meatus.

GRANT, Philadelphia.

CONGENITAL ATRESIA OF THE ESOPHAGUS: A STUDY OF THIRTY-TWO CASES. THOMAS H. LANMAN, Arch. Surg. **41**:1060 (Nov.) 1940.

This condition is discussed in detail, and 32 cases are reported. Of particular interest are the characteristic and striking symptoms. "They are attacks of choking

and cyanosis occurring in the first few hours of life, and almost invariably these attacks are aggravated whenever any fluid is offered the infant. Attempts to swallow fluid are followed by immediate regurgitation and are usually accompanied with evidence of aspiration of fluid into the air passages, shown by choking, coughing and cyanosis." These symptoms should suggest the diagnosis. A roentgenogram is helpful, but it must be made without the use of a contrast medium, as frequently a fistula is associated with the atresia, so that the giving of barium sulfate by mouth is accompanied by great danger.

All 30 of the patients who were operated on died. The author feels, however, that improved technic and greater experience will eventually result in a successful outcome of direct anastomosis of the esophagus.

KAISER, Rochester, N. Y. [AM. J. DIS. CHILD.].

THE PATHOLOGIC PHYSIOLOGY OF BRONCHIAL ASTHMA IN CHILDREN (WITH REFERENCE TO THE ROLE OF INFECTION). HENRY N. PRATT, New England J. Med. **223**:626 (Oct. 17) 1940.

The fundamental pathologic physiology of all atopic and anaphylactic phenomena is increased capillary permeability, smooth muscle spasm and excessive mucus secretion.

The part played by infection may be divided into four categories: bacterial allergy; infection of the respiratory tract acting as a trigger mechanism; infection secondary to the asthmatic attack, and focal infection. It has often been predicated that a child may become sensitized to bacteria which he harbors somewhere in the respiratory tract. The occasional satisfactory result following therapy with autogenous or stock vaccine is in most cases due to shock and is entirely nonspecific.

Infection of the respiratory tract, such as the common cold, often precipitates an asthmatic attack in an atopic child. The child is usually sensitive to some food or inhalant which he can tolerate except in the presence of infection. This has been clearly demonstrated over and over again by abolishing the extrinsic factor; the child then has a cold without asthma.

Infection may be secondary to an attack of asthma initiated by some extrinsic factor to which the child is sensitive. Such an infection probably originates in the inadequately aerated parts of the lungs. Once established, it may produce a chronic irritation in the bronchi which causes the attacks of asthma to continue intermittently for several weeks.

Focal infection seldom plays a part in childhood asthma. In an allergic child the lymphoid structures in the upper respiratory tract tend to hypertrophy, so that one is sorely tempted to remove the large, boggy tonsils and adenoids. Statistically it has been shown that removal of these structures has no lasting effect on the asthma.

GEGENRACH, Denver [AM. J. DIS. CHILD.].

LIMITATIONS OF GASTROSCOPY. I. R. JANKELSON and C. W. MCCLURE, New England J. Med. **224**:64 (Jan. 9) 1941.

Gastroscopy, like any other method of endoscopy, has distinct limitations. The authors present this paper to call attention to these and to help in the proper evaluation of the clinical use of the gastroscope. The paper is based on 800 cases observed over a period of three and one-half years. In all cases the usual roentgenographic routine was employed in diagnosis. The gastroscopic and the roentgenographic findings were then compared.

Any serious disease or obstruction of the esophagus is a contraindication to gastroscopy. The procedure is frequently impossible in cases of severe curvature or arthritis of the spine. Severe cardiac disease and aneurysm of the aorta, as well as uncontrollable cough or dyspnea, are contraindications. The authors do not perform gastrostomy in cases of acute gastric hemorrhage. The intelligent

coordination of roentgenographic and gastroscopic examination will diminish the error in diagnosis of gastric disease. Gastroscopy is limited in its field of usefulness, whereas roentgenography has wide scope in determining the morphologic appearance of the digestive tract. Failure of the gastroscope to visualize changes in the stomach does not exclude the presence of intragastric disease; on the other hand, such visualization may be difficult of interpretation. The differential diagnosis of ulcerating carcinoma and gastric ulcer is not always possible. The accuracy of gastroscopic study depends to a large extent on the experience of the observer and his ability to coordinate the available gastroscopic, roentgenographic and clinical evidence.

SCHALL, Boston.

HEMORRHAGE IN OTOLARYNGOLOGY: METHODS OF CONTROL. MARVIN F. JONES, New York State J. Med. **41**:580 (March 15) 1941.

This article contains the substance of lectures given before the Oregon Academy of Otolaryngology in April 1940. Jones reviews the anatomy and the blood supply from a surgical point of view and then describes in detail the management of hemorrhage from the tonsillar fossa, the adenoid area, the septum, the sphenoid sinus, the maxillary sinus, the ethmoid labyrinth and the frontal sinus, as well as "general nasal hemorrhage." He then discusses aural hemorrhage and concludes with a few comments on hemorrhage from the larynx.

Jones mentions the frequency of nasal hemorrhage in adolescence but does not give a cause, unless it be "glandular maladjustment." If bleeding is troublesome during submucous resection, it is probably from the artery in the nasal crest. This can be closed by applying a blunt instrument and tapping with a mallet. He thinks that petrolatum gauze is the dressing of choice. Bleeding during operations on the ethmoid sinus performed with local anesthesia may be troublesome but is controlled by pressure plugs of gauze soaked in hemostatic solutions. External operation admits tying off the anterior ethmoid artery. Bleeding after tonsillectomy is best prevented by making sure both fossae are dry before sending the patient back to bed. In cases of general nasal hemorrhage Jones has discontinued testing for coagulation and bleeding time save when hemophilia is suspected. Aural bleeding in cases of fracture of the skull should be left severely alone, lest one by meddling induce meningitis. Bleeding from the sigmoid sinus and the emissary vein during mastoidectomy is readily controlled by pressure. This article is an excellent résumé of an important subject.

VOORHEES, New York.

A NEW TREATMENT FOR HAY FEVER. ERNEST J. ELSBACH, New York State J. Med. **41**:1248 (June 15) 1941.

Elsbach observes that allergic rhinitis is common in the United States, that it causes great loss of working time in industry, that it has many "remedies" which are of little use, that cutaneous tests are often troublesome and inconclusive and that such "inoculations" as are used must be repeated each year, or oftener, and cannot be made during the attack.

Elsbach offers a "new treatment," which is new only in this country, since it has been used with success in Europe for some years. He believes that "some pathologic condition of the vegetative nervous system" is at fault. There are, of course, grass pollens, food allergens, ultraviolet ray activity, natural (sunlight) or artificial (quartz–mercury vapor lamps), and psychic factors (fright, fear, anxiety). When pollen albumin causes allergic rhinitis, protein therapy properly administered seems logical. Elimination of known basic diseases, e. g., intestinal stasis and sequelae, the use of strontium and calcium to diminish irritation of the sympathetic nervous system, etc., have a place, but more dependence can be placed on treatment with the "products of metabolism of bacillus coli." Pure cultures of the bacilli are incubated at body temperature, transplanted, heat killed, diluted with saline solution, filtered and prepared for injection. Eight to twelve intramuscular

injections of 2 cc. each are given—one a day for five days—and the injections then repeated at chosen intervals for two or three weeks. No severe reactions or bad results ensued. The ages of the patients ranged from 9 to 56 years.

VOORHEES, New York.

BODY SECTION ROENTGENOGRAPHY AS A DIAGNOSTIC AID TO THE OTOLARYNGOLOGIST. S. MOORE and A. J. CONE, Surg., Gynec. & Obst. **72**:514 (Feb. 15) 1941.

Superfluous images greatly limit the usefulness of roentgenograms, and this applies particularly to those taken of the upper respiratory tract. A new technic of using a coordinated, synchronized movement of the x-ray tube and film about a given point will disperse unwanted shadows to a remarkable degree and make a selected level stand out in clear detail. As applied to the field of otolaryngology, the technic is a valuable adjunct to conventional roentgenograms and finds its greatest usefulness in outlining the size, position, extensions, anomalies and pathologic states of the ethmoid and sphenoid sinuses. In cases of congenital absence of the auricle, the extent of the external auditory canal can be clearly demonstrated. In cases of suspected tumor of the auditory nerve detailed visualization of the internal auditory canal is possible. Studies of the larynx and temporomandibular joint by this method yield information about pathologic processes hitherto impossible to obtain with the usual roentgenograms. There is undoubtedly a place for this technic in many cases in which the diagnosis is obscure and special studies are indicated.

HENRY A. MILLER, Philadelphia.

MISUSE OF TANNIC ACID. Editorial, Lancet **2**:627 (Nov. 16) 1940.

When tannic acid was hailed a few years ago as the perfect dressing for burns, there seemed no pitfalls to avoid, no circumstances in which its use could prove disastrous. Yet at a recent meeting of the Royal Society of Medicine all speakers agreed that tannic acid must never again be used for burns of the face. Their experience has indicated that such burns should be treated by skin grafts as soon as the surfaces are adequately prepared by saline baths. Sulfanilamide and its derivatives are proving useful in the local treatment of infection. In the navy and air force a jelly containing 1 per cent gentian violet and merthiolate (1 : 5,000) has now been substituted as a first aid dressing.

H. A. MILLER, Philadelphia.

USE OF HISTAMINE IN TREATMENT OF NEUROVASCULAR HEADACHE. W. L. REID, M. J. Australia **2**:307 (Oct. 5) 1940.

Reid gave small repeated subcutaneous doses of histamine to 5 patients with the neurovascular type of headache. One patient experienced only partial relief, while the other 4 obtained complete relief. Each patient, after the possibility of an intracranial pathologic lesion was excluded and the reaction to an intradermal test injection of histamine was found to be positive, was given graduated doses of histamine in the following manner: The solution used consisted of 1 mg. of histamine dissolved in 10 cc. of physiologic solution of sodium chloride. On the first and second days 0.05 mg. of histamine (0.5 cc. of the solution) was injected subcutaneously twice a day, and on the third and fourth days 0.075 mg. (0.75 cc. of the solution). Thereafter, up to the end of the third week, 0.1 mg. (1 cc. of the solution) was given twice a day. When this course was finished, each patient was given one injection of 0.1 mg. of histamine once a week. If the patient remained free from symptoms during the next two or three months, an attempt was made to decrease the dose by spacing the injections. If the symptoms recurred after the dose was diminished the injections were again increased to one a week. The

author applies the term "neurovascular headache" because there is fairly conclusive proof that the aura and pain in many complaints of head pain are due to a disturbance of the neurovascular mechanism controlling certain intracranial and extracranial blood vessels. Of the various patterns of this group, migraine is perhaps the best known. There are several other patterns which have a neurovascular mechanism, and some of these are as common as the classic migraine. It is emphasized that the use of histamine must not be regarded as a cure-all for every type of headache. Each case must be thoroughly investigated, and only when all possibility of a pathologic lesion has been excluded should a neurovascular mechanism be considered.

J. A. M. A.

THE INCIDENCE OF HAEMOLYTIC STREPTOCOCCI IN THE THROATS OF OBSTETRIC NURSES: RESULTS OF TREATMENT OF TONSILLECTOMY AND SULFANILAMIDE. J. N. CHESTERMAN and S. SCANDRETT, M. J. Australia **2**:695 (Dec. 28) 1940.

The authors' study shows that nurses working in an obstetric hospital to which is attached an isolation block for the treatment of puerperal infections run considerable risk of acquiring hemolytic streptococci in the upper respiratory tract. This risk is greatly increased by the presence of tonsillar tissue. Tonsillectomy proved successful in eliminating the infection from the throat in the majority of instances. When tonsillectomy alone was not enough, sulfanilamide overcame the infection. However, sulfanilamide was not effective in the presence of pronounced abnormality of the upper respiratory tract.

GONCE, Madison, Wis. [AM. J. DIS. CHILD.].

ASTHMA REFLEX ORIGINATING IN THE TONSIL. F. CHAVANNE, Oto-rhino-laryng. internat. **24**:257 (Oct.) 1940.

In 1912 Chavanne reported a case of asthma in a child aged 14 years which had existed for twelve years and which was relieved immediately by the removal of a papilloma of the uvula. In 1929 a second case was observed. The patient, aged 7½ years, had had monthly attacks of asthma since the age of 2 years. No abnormality was discovered except hypertrophied palatine tonsils. Complete relief was obtained by tonsillectomy.

In both cases the asthma reflex was of purely pharyngeal origin, without nasal relay or concomitant nasal lesion. The reflex in the first case was dependent on the pneumogastric nerve, and in the second the trigeminus nerve was responsible. The observations are important in indicating the possibility of a pharyngeal origin of asthma. Cases of this type are rare. The majority of cases of asthma belong in the realm of general medicine.

DENNIS, San Diego, Calif.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY, INC.

ERNEST M. SEYDELL, M.D., *President*

ABSTRACTED BY ALFRED LEWY, M.D.

Seventy-Fifth Annual Meeting, Atlantic City, N. J., May 28-29, 1942

(Concluded from Page 143)

Studies of Deafness in Twins: Otosclerosis in Identical Twins; Three Case Histories. DR. EDMUND P. FOWLER, New York.

There is no satisfactory theory as to the cause of otosclerosis, and the so-called cures have failed. Even though the otologist may be able to prevent or control otosclerosis, its inheritability will remain unchanged and so the problems of prevention, arrest and alleviation will always be present.

Identical twins are subject to identical hereditary influences. They react similarly to many biologic tests and inherit strong tendencies to acquire many diseases and disorders at similar age periods, but there are always some differences in the environment. Only 6 instances of otosclerosis in identical twins have heretofore been reported.

Shambaugh Sr. and Shambaugh Jr. have reported 2 instances of nerve deafness without ankylosis of the stapes in identical twins. I have not included these in the 6 instances. Shambaugh Jr. reported three pairs; one pair in similar environment showed a difference of sixteen years in time of onset, with less deafness in the one who had fewer colds; and one pair whose otosclerosis began five years earlier in the one who had more head colds; in another pair the otosclerosis began five years earlier in one and progressed with episodic infections of the upper respiratory tract. The second twin was much deafer after pregnancy. Hagens reported one pair with a few years difference in the time of onset; in one of these the impetus was attributed to childbirth. In regard to the three pairs I am reporting I have considered the blood count, the chemical components of the blood, the Wassermann reaction, the basal metabolism, the fingerprints, the photographs, the eyegrounds, the teeth, the electrocardiograms and repeated audiograms. The case histories are set forth in great detail.

Only in the case of the third pair of twins was there an impressive history of deafness in the family. The first pair had hypertrophied lymphoid tissue in the pharynx, the deafer one showing a greater amount. In the second pair, the deafer twin had fewer head colds. Both had hypertrophied lymphoid tissue in the nasopharynx. In the third pair, the deafer twin had more head colds and hypertrophied lymphoid tissue and one earache at 5 years of age. The better hearing twin, with fewer head colds, had a slight involvement of the lung and pleura, now healed. In the second and third pair of twins there was greater deafness in the one who drank less milk, but in the first pair the deafness was greater in the one who drank more milk. In each instance the twins slept in the same bed, but in the first and third pairs the deafer twin slept near the window, which may have accounted for more lymphoid tissue in these two persons.

Menstruation and child bearing caused no notable changes in the hearing acuity except in twin "A" of the third pair; during her menses she was nervous. Although

frequent head colds would appear to coincide with more severe deafness in the cases of otosclerosis reported to date, it must be kept in mind that this increase in severity may have been due to superimposed deafness and not to the otosclerosis per se. Also susceptibility to colds is not necessarily only environmental. It is often inherited. Other things besides head colds affect hearing in persons with otosclerosis. It may be that men are less subject to otosclerosis than women because of their relative freedom in adolescence and later from readjustments of the endocrine mechanism. Many other deafnesses noted may have had various underlying causes.

All of these observations were made after the otosclerosis was well established. What is needed is careful investigation before the beginning of otosclerosis. Otologists should persist in making early and regular examinations of children, especially of identical twins, of otosclerotic ancestry.

I have collected 150 twins and triplets who now show no deafness. Those with family histories of otosclerosis have been kept under close observation for many years. They will also be used in the development of tests for early diagnosis. There is now no sure test, either for early or for late otosclerosis.

In addition I have carefully examined 40 pairs of twins who have various types of deafness. These statistics may appear futile. It does not mean that they are worthless. The data on otosclerosis can be compared with data on other forms of deafness and some day something worth while will be discovered.

I again appeal to you for names and addresses of identical twins one or both of whom have partial deafness or whose parents have otosclerosis.

DISCUSSION

DR. CHARLES B. DAVENPORT, Cold Spring Harbor, N. Y.: Genetic and environmental factors are constantly interacting.

To determine how far the pathologic picture and progression of otosclerosis are due to genetic factors and how far to environmental factors cooperating therewith, it is desirable to study pairs of identical twins, because in them the genetic factors are probably the same. That part of the pathologic picture which is the same is probably due to genetic factors. Environmental factors may act indirectly through other links in the chain. A may affect B in the organism, B affect C and C affect D, which is the thing one sees.

So far as environment has been different for identical twins, their pathologic pictures may differ. In identical twins who are both otosclerotic, this alteration would be due to genetic factors. The open window, the smoking habits, swimming, child bearing and so on will also have their influence.

Dr. Fowler's project of getting at the causes of the modifications in the progress of otosclerosis that are due to nongenetic factors has been well planned and may be expected to yield results.

STACY R. GUILD (Ph.D.), Baltimore: I feel that Dr. Fowler's plan of following large numbers of identical twins is the only method by which it will be possible to establish the difference between the effects of heredity and those of environment. The small number of cases reported so far is not sufficient. The effort to get these twins under observation before the age of onset is commendable. The age of onset is, of course, a matter of the subjective impression of the patient. I congratulate Dr. Fowler on the completeness of his data.

One must remember that persons with the same environment for both ears may have unilateral otosclerosis, histologically or clinically. This makes me a skeptic.

Another thing—a 0.5 mm. difference in position of the margin of the otosclerotic area adjacent to the oval window may make the difference between the presence or the absence of clinical symptoms.

Emphasis has been placed on the difference in susceptibility to colds. Why did one twin have more colds than the other? Was this a development from the same soil or were the increased susceptibility to colds and the earlier age of onset due to a common cause?

The influence of pregnancy is an old problem. In a case reported by Shambaugh, I believe, a twin who had a child was eight years later than the other in showing symptoms of otosclerosis. In another reported by Dr. Hagens, the reverse was true. I do not believe that problem will be settled until some one carries through a program with women from otosclerotic families as Dr. Fowler is doing with twins. There will have to be 500 girls from otosclerotic families followed from childhood on through the child-bearing age, with 500 others as controls. Then one may see what the average histories have been in the final results.

DR. F. R. SPENCER, Denver: Many pieces of research will not be important at the time but may acquire importance later. I should like to encourage Dr. Fowler in his work, and I hope he will find something helpful to otologists.

DR. EDMUND P. FOWLER, New York: I do not think two ears are in the same environment. Even in the basic inheritance, the right hand may inherit a tendency different from that of the left.

Neurologic Complications of Serum Sickness, with Special Reference to the Ear. DR. H. MARSHALL TAYLOR, Jacksonville, Fla.

Foreign serums administered parenterally may affect adversely both the somatic and the sympathetic nervous system. Numerous syndromes may follow their use. The symptoms usually subside, but there may be residual deafness.

Since 1908, when this disease was first described, reports have accumulated in the literature of nearly all countries, especially that of France. The first case reported in the United States was that recorded by Richardson in 1917.

Most frequently serum sickness follows the administration of antitetanus serum, which is given more often to adults, in whom neurologic complications are more likely to occur. The relative rarity of cases in which it follows the injection of diphtheria antitoxin is believed to be due to the comparative immunity of children. According to one theory, the primary site of the disorder is the blood vessels; according to another it is the cells. Postmortem observations and laboratory evidence indicate that the process includes vasodilatation and edema of the skin, muscles, bursas and tissues in the joints. It may cause nutritive impairment of nerve tissues and interfere temporarily with their activity by occasionally causing cell death, according to Kraus and Chaney. These authors noted that the vascular disorder produced a meningeal disturbance and excessive spinal fluid. They concluded that "an edematous process may occur in the perineural sheaths, or the nutrition of the roots or nerves may be interfered with by impairment in the blood supply, thus causing various radicular and neural syndromes."

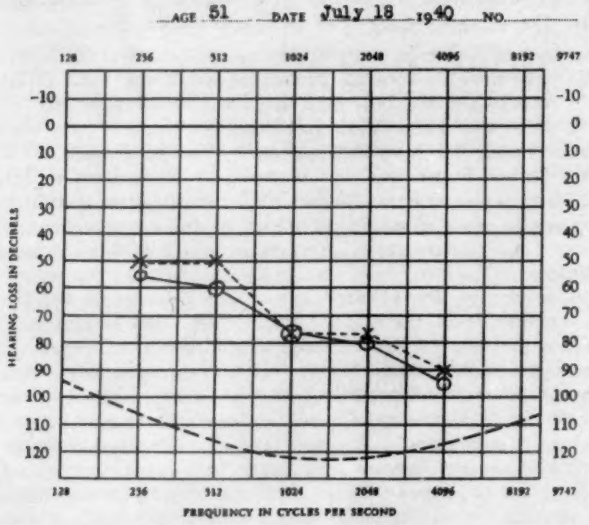
The most frequent neuritic occurrence in the reported cases was the involvement of the suprascapular nerve combined with involvement of the axillary or long thoracic nerve, all from the fifth, sixth and seventh cervical nerves. The involvement of the upper brachial nerves remains unexplained. Since the fifth and sixth cervical nerves are most frequently affected in cases of paralysis of the brachial plexus, it may be that the anatomic explanation lies in their position. They come from vertebral foramens higher in the neck than do the others; consequently they traverse a greater distance to the axillas, and in their journey they pass through the fasciae of the neck between the anterior and middle scalene muscles. As they swell with edema, the resulting pressure against these structures may bring about ischemia of the nerves with consequent paralysis.

There may be meningeal, spinal and cerebral lesions. In the cerebral form, the cranial nerves may be involved.

In Doyle's series the average interval between the injection of serum and the onset of serum disease was six and eight-tenths days. Between the onset and the appearance of neurologic symptoms there was an average interval of two and one-tenth days. The symptoms are usually pain, fever and urticaria; in addition, headache, nausea and vomiting may occur. Swelling of the lymph nodes and joints is also sometimes noted. Severe neuralgia may be present, and the location bears no relation to the site of injection.

Features of the cerebral syndrome are meningeal irritation, choked disks, alexia, hemianopia, aphasia and hemiplegia. Spinal fluid pressure is increased, and the cellular reaction is slight. Paralysis of the cranial nerves or a bulbar syndrome may occur. Isolated involvement of the axillary, ulnar, median, radial, external popliteal or entire sciatic nerve has been reported. Optic neuritis with edema of the retina, swelling of the disks, ptosis, strabismus, diplopia, nystagmus, facial paralysis, huskiness of voice, tinnitus, deafness, hemiplegia, paraplegia and pseudotabes have all been reported.

The prognosis is usually good, although complete restoration may require months. On the other hand, partial or complete residual paralysis may remain. Treatment in the acute stages consists of medication with atropine and epinephrine and dehydration, with intake of the fluids restricted and with retention enemas of magnesium sulfate. Spinal drainage is carried out. Large doses of caffeine and intravenous injections of dextrose are useful; later electrotherapy and massage are given in cases of muscular paralysis.



Curve of hearing loss in patient with serum sickness from 5,000 units of tetanus antitoxin (air conduction: right ear o, left x).

Aural Manifestations.—Involvement of the eighth nerve occurs infrequently, and its rarity emphasizes the importance of reporting it. Amberg and Hewitt in 1935 reported a case of inner ear deafness following the injection of 120,000 units of tetanus antitoxin. The deafness was total and permanent. In 1936 Cutter reported a similar case with improvement; the loss of hearing was 69 per cent on one side and 61 per cent on the other, which improved to 21 and 9 per cent, respectively. McCready in 1937 reported a case of extreme dizziness occurring nine days after the injection of 3,000 units of tetanus antitoxin; five days later there was a considerable degree of bilateral deafness; the hearing loss was 46 per cent on the right and 45 per cent on the left; the hearing did not improve. This condition was interpreted as diffuse serous labyrinthitis.

My own case was as follows: A man aged 51 years was given 5,000 units of tetanus antitoxin intramuscularly for a laceration of the forefinger. Five days later he was awakened by an attack of urticaria with pronounced swelling of the lips, ears and scalp. Six hours later there were intense ringing in the ears and

rapidly developing deafness. An otologist was immediately called in consultation. The patient claimed that the hearing was previously normal, a statement which was corroborated by the family and the employers. There was no evidence of previous disease of the nervous system. Seven years prior to that time, however, he had received tetanus antitoxin. The Wassermann and Kahn tests were negative; the physical examination showed nothing abnormal. I saw the patient five months after the onset of the deafness. The drum membranes were normal. He could hear loud conversation at 4 feet; tuning forks c-4 and c-3 were heard with effort in both ears. By bone conduction fork aI was heard for five seconds, compared with the normal of twenty seconds. The audiometric examination showed loss of 68 per cent in the right and 70 per cent in the left ear, with the greatest loss of hearing for high tones. The eustachian tubes were patent, and caloric tests produced no reaction; ten turns to the right produced horizontal nystagmus to the left of thirty-five seconds' duration; ten turns to the left, a nystagmus to the left of forty seconds' duration. These tests caused slight vertigo.

Repeated tests during the next nine months showed no change in the audiometric findings. The diagnosis was nerve deafness that appeared to be permanent.

The permanent perception deafness appears to be without doubt the direct result of the administration of tetanus antitoxin. Whether it was caused by diffuse serous labyrinthitis or by compressive neuritis similar to that of Bell's facial paralysis has not been demonstrated. There is a question of previous sensitization occurring seven years ago. It may be well when serum is indicated after a previous injection, even one made years ago, to use bovine serum. Deafness may have been due to the serum given in treatment rather than to pathologic changes due to meningitis. This may also be true with diphtheria and scarlet fever when the middle ear is intact.

DISCUSSION

Dr. James A. Babbitt, Philadelphia: I recall with appreciation two brilliant, somewhat pioneer pieces of work by Dr. Taylor: One was a study of the anatomic variations in water-accustomed animals and the physiologic effects of long exposure to cold in swimming. The second was an investigation of the prenatal effects of faulty administering of quinine and other drugs to pregnant mothers. He has now ventured into another somewhat unexplored field, the possible otologic complications of serum sickness.

The literature on serum sickness has been meager, particularly that from the otologic standpoint. Conference with four leading pathologists in Philadelphia and many neurologists and internists failed to elicit any knowledge of otologic complications in serum sickness.

Granting the effect of nutritional impairment in the nerve tissue from edematous processes of nerve sheaths, analogous to the neurorecidive reaction in parasyphilis, which has been carefully eliminated in the author's case, have other causes such as poisons, toxins and infectious diseases been ruled out?

With the comparative absence of vestibular symptoms in this case of sudden deafness, such as were recorded in other cases, could this condition be indicative of blood collections of a thrombotic type or a lesion in the upper neural centers? Dr. Taylor did not see the patient until five months after the onset, and earlier symptoms may have been overlooked.

Isn't it unusual that so few of the other more customary neurologic complications of serum sickness have occurred, and what is the explanation? If the auditory or optic nerves are to be singled out, would it not be valuable to have the mechanism clarified?

Dr. Taylor has noted the comparative immunity of children to neurologic complications, thus releasing the danger of diphtheritic antitoxin. His observation of low conduction by bone corresponds with my recent observation in a case of acute herpetic involvement of the middle ear, the facial nerve and the labyrinth of the type described by Ramsay Hunt. Prophylactic precautions should be established in administering serum.

ALFRED LEWY, Chicago: Among the many reported cases of neurologic damage resulting from the use of serums for curative or preventive purposes, those concerning the ear are scant. By far the largest number concern the brachial plexus, but cases of optic neuritis and cases of general encephalopathy are not wanting.

Dr. Archibald Hoyne, in charge of the Municipal Contagious Disease Hospital in Chicago, and Dr. E. K. Musson, chief of the communicable disease section of the Chicago department of health, have no knowledge of cases affecting the ear, nor does Dr. Béla Schick, of New York (personal communication).

Alexander in Pfaundler's "Handbuch der Pediatrie" does not attribute any aural disturbance to serum sickness, nor is the ear mentioned in the chapter on serum sickness.

In spite of the current theory that Ménière's symptom complex is often due to allergy, aural disturbance attributed to serum or vaccine has rarely been noted. Perhaps it has been masked by other allergic manifestations or by the condition of the patient.

DR. NORTON CANFIELD, New Haven, Conn.: I wish to give a follow-up report on a case reported by Dr. McCarthy in 1937. That child has had no return of function and is now in a school for deaf children.

If the actual lesion is in the otic capsule, there are possibilities for treatment. If the hearing rapidly decreases over a period of some hours, the lesion, I think, may be either inside the otic capsule or along the course of the eighth nerve.

Reducing the tissue fluid locally has been effective in some instances, and I think one should consider prompt lumbar puncture as a means of reducing the labyrinthine pressure. The intravenous injection of hypertonic solutions should also be considered, but in a case of rapidly decreasing hearing I would not hesitate to decompress the labyrinth by doing a fenestration operation on the semicircular canal.

DR. ERNEST M. SEYDELL, Wichita, Kan.: Dr. Taylor spoke to me about this case a year ago. I had 2 cases which I thought were due to this, in the twenties, and I think I found a report of a case in the *Monatschrift für Ohrenheilkunde* but was unable to locate the report again.

DR. EDMUND P. FOWLER, New York: Every one has seen patients with mumps who underwent total destruction of hearing and labyrinthine function. There is no direct connection between the parotid gland and the labyrinth except theoretically via the facial nerve. If the sheath of that nerve is involved, there may be no symptoms, as it is tough, but the more delicate eighth nerve may be affected. This is only a theory, but the otologic complication does not come on as with the symptoms of serum sickness. It always seems to be on the side of mumps, and I should like to have Dr. Taylor in his studies think of other viruses. Mumps may be a virus disease. There may be local meningitis afterward. Perhaps virus must also be considered in serum sickness.

DR. FRENCH K. HANSEL, St. Louis: Among the organs affected in shock in man the auditory nerve is one of the most infrequently involved. With the present day tendency to use toxoids instead of antitoxins, otologists will see less trouble from serum disease. It will be interesting to see what happens later, when otologists will be using principally one type of sulfonamide compound. The incidence of serum disease is higher in cases in which a large amount of serum is used.

In diagnosing the allergic type of Ménière's disease one usually finds it associated with some other manifestation of allergy. In regard to treatment I would suggest use of large doses of histaminase. In the cases of acute angioneurotic edema or of urticaria of all types it is difficult to relieve the edema. Early injections of epinephrine hydrochloride or intravenous injections of theophylline with ethylene diamine U. S. P., which is used for severe asthma, tend to cause elimination of large amounts of fluid.

DR. H. MARSHALL TAYLOR, Jacksonville, Fla.: I am not surprised that the discussers have not reported a volume of these cases. Some one else reported this case and I have never observed a case like it. This patient was first seen by an

otologist in my community. In view of the enormous edema and urticaria that the patient had, it was almost impossible to escape the conclusion that the deafness was the result of the perineural swelling.

In 1939 Bennett reported 115 cases of serum sickness, and six discussers reported additional cases of involvement of the brachial plexus. There were 122 cases in all.

Encephalography in Otogenic and Rhinogenic Complications. DR. HANS BRUNNER, Chicago.

Air was first injected into the ventricles of the brain in an attempt to diagnose different forms of hydrocephalus and tumors of the brain. Later the procedure was extended to inflammatory diseases of the brain. Air can be injected directly through the skull or through the atlanto-occipital membrane and also by lumbar puncture. I have used only the latter two routes.

The technic I have employed for lumbar encephalography is the one now essentially in vogue. Premedication with phenobarbital and morphine is used. The use of morphine has been objected to by some, who advised substituting atropine sulfate. As a precaution, some physicians administer extract of adrenal cortex thirty minutes before injecting air. General anesthesia is contraindicated. It is desirable to begin the operation in the x-ray room, but the operation may be done in one room and the patient taken to the x-ray room after the injection of air. Spinal puncture is done and the pressure of the cerebrospinal fluid determined while the patient is in the horizontal position. The patient then sits up slowly, with the head moderately flexed. The fluid should be withdrawn slowly and a lesser quantity of air slowly introduced. Clinicians differ as to the quantity of air to be injected and the ratio to the amount of fluid withdrawn. I prefer removing 5 cc. of fluid, then injecting 3 cc. of air, then again withdrawing slowly 10 cc. of fluid and injecting 6 to 8 cc. of air.

If the flow of fluid stops too soon, the needle should be slightly changed in position or the head bent backward. If cardiac collapse appears, the patient should be placed in a horizontal position without disturbing the needle and stimulants administered. This should also be done if there is a complaint of nausea. In the presence of respiratory embarrassment, the needle must be withdrawn and oxygen administered by inhalation without delay.

Once air has been introduced, roentgen ray exposures must be made at once. For the otolaryngologist the rays should be focused anteroposteriorly, posteroanteriorly and laterally (stereoscopic). It is also desirable to repeat the roentgenologic studies after twenty-four hours.

Encephalography is not without danger. The reported mortality rates vary from 0.7 to 12 per cent. Some patients suffer with headache, emesis, chilliness, diaphoresis, restlessness, backache and circulatory disturbance with pallor. To these symptoms should be added paresthesias, mental confusion and pains in the thorax and abdomen. The symptoms may last up to six days, especially in cases of tumor of the brain. This shows the need of holding the amount of injected air below the point of tolerance. Occasionally there are a rise of body temperature and leukocytosis, with increase in globulins in the spinal fluid and stasis in the eyegrounds. These signs disappear in a few days unless caused by the intracranial complication itself.

In the practice of the otolaryngologist this diagnostic procedure will be seldom needed. The contraindications may be summed up as follows:

1. Coma.
2. Pronounced disease of the heart or of the aorta.
3. Senility or infancy.
4. Acute respiratory infections, open tuberculosis and furunculosis.
5. Increased intracranial pressure with choking of the disk of more than 2 diopters or lumbar pressure beyond 300 mm. of water in the sitting position or 200 in the lying-down position. In such cases there is danger of herniation of the medulla, and it is better to resort to ventriculography.

6. Suspected complications in the posterior fossa. I have violated this contraindication in 2 cases, but the air did not enter the ventricles.

Lumbar encephalography may be indicated in the following conditions: abscess of the temporal lobe, abscess of the frontal lobe, malignant neoplasm of the temporal bone and of the paranasal sinuses, and complications suggesting cerebral abscess.

An abscess of the temporal lobe is indicated by the following changes: compression of the ventricles on the affected side; dilatation on the normal side; lateral displacement of one or both ventricles, especially the anterior part of the ventricles, toward the unaffected side; diminished filling of the subarachnoid spaces of the convexity on the affected side.

Neurosurgeons claim that lumbar encephalography may produce diffuse meningitis. I could not find record of a single instance actually proving that danger to be present in cases of chronic or acute abscess of the brain, and it has not occurred in my experience. Theoretically, I believe that lumbar encephalography is no more dangerous than simple spinal puncture. This does not imply rejection of ventriculography in these cases, but that is a more complicated surgical operation and also involves risks.

An abscess of the frontal lobe is manifested by compression of the anterior horn on the affected side and by dislocation of the ventricles on that side backward and toward the normal side.

A malignant tumor of the temporal bone and paranasal sinuses may not produce definite neurologic symptoms. In many cases of carcinoma of the right middle ear, both ventricles were displaced to the left, and the superior and the lateral edge of the right ventricle were elongated. The third ventricle was dilated, the right inferior horn compressed and the subarachnoid spaces of the convexity better filled on the left than on the right.

When a suspicion of an abscess of the brain exists, encephalography may prevent unnecessary exploration. It is required to clarify the diagnosis in the following conditions:

1. Otitic hydrocephalus (I prefer the term toxic hydrocephalus). The subarachnoid spaces of the convexity are well filled only on the side that is normal, as in many of my cases. The basal cisterns may be normal or enlarged. The ventricles are normal, relatively small or slightly enlarged. The clinical syndrome of toxic hydrocephalus may be due to internal hydrocephalus or to edema of the brain. (The pathologic changes characterizing internal hydrocephalus can also be produced by intense application of roentgen rays to young animals.) These findings differ from those in cases of cerebral abscess and aid in distinguishing an abscess of the brain from toxic hydrocephalus.

2. Intermittent purulent meningitis. This affects the convexity of the brain more than the base and is characterized by symptoms referable to the convexity and by the chronic intermittent course. In 1 case I obtained normal encephalograms by suboccipital puncture.

3. Osteomyelitis of the skull. When symptoms resembling those of an abscess of the brain develop, encephalography is of great help. In 2 cases I observed normal encephalograms and thereby prevented useless puncture of the brain. Lumbar encephalography is more advisable than ventriculography involving injury of the skull.

This is by no means an extensive study of encephalography. The material at my disposal is too small. It is my aim to stimulate the interest of otolaryngologists in the subject.

Electroencephalography in Relation to Otology. Dr. Robert A. Schwab and Dr. Richard A. Carter, Boston.

When Prof. Hans Berger published his first communication, the result he described was considered an artefact. This view was ruled out by Professor Adrian, who established a complicated laboratory for this purpose. Professor Adrian had obtained the same result from the British water beetle. Here are two

slides showing the similarity between Professor Adrian, winner of the Nobel Prize, and the British water beetle.

The waves from the normal human brain are the alpha and the beta. The alpha waves disappear when the light is turned on and return when it is turned off. When the whistle blows, there is hardly any change in the rhythm. We have found the clinical application of this test to be limited. There are three types of abnormal activity. First there is the slowing of the rhythm from 10 per second down to 3 or 6 per second, with a greater build-up in the voltage. These waves, about five or six times higher in voltage and much slower than normal, are called delta type and are found in many clinical conditions, such as disease of the brain and deprivation of oxygen.

The second abnormal type is a fixed frequency between 16 and 22 per second. The third type is a mixture of the first and second types. This consists of a three cycle wave, the delta wave and the fast wave and is found nearly always in epilepsy and can be used to diagnose petit mal.

A slide was shown, taken from an infiltrating glioblastoma of the parietal region. A similar one was taken from the brain of a patient with an acute attack of vertigo from Ménière's syndrome. One way to use these waves in localization would be to put on more electrodes and keep moving them around to locate abnormal activity or activities. A better way, and the one we use, is to find where the slow waves are out of phase.

In this case of hematoma (slide shown) the activity comes from a part of the brain which has been deprived of oxygen or of sugar. Pathologic processes do not give off electric discharges. It is the surrounding brain which is having its normal metabolism interfered with. If one can find places where the waves are opposite each other one can see that the source is between these two large waves and that the disturbance must come from the electrode between them. We call that a "phase shift," and it is the means we use in localizing the process.

[A number of slides were shown to illustrate the findings and results.]

One slide shows the disappearance of abnormal waves one month after successful removal of the lesion. The next demonstrates a definite electrical focus and is called positive localization. With a definitely abnormal electroencephalogram, localization may nevertheless be doubtful.

In the A group of cases, in which we felt certain of the localization, we were shown in the follow-up study by operation or postmortem examination to have been 85 per cent accurate. In the B group, which were the doubtful ones, we were 55 per cent accurate. The total accuracy was 72 per cent. In the C group, which did not show any pathologic condition, the follow-up revealed that 91 per cent of the patients had no intracranial lesion. We compared the 500 localizations with the results of other forms of determining localization.

In neurologic examinations alone our senior neurologist was able to strike an average of 71 per cent. With roentgen diagnosis the average was 20 per cent.

Air studies were 90 per cent accurate in localization of intracranial lesions. In cases in which it is suspected that cerebral abscess is complicating sinal or aural disease, if the apparatus is available, it is useful for two reasons: The examination is painless. It is no more complicated than making an electrocardiogram. Operation on or injection into the patient is not necessary. If an abnormal record is obtained, even without localization, it is strong evidence of intracranial disease. We have made the encephalographic examination in the operating room after exposure of the brain and believe the idea to be new. We can record nystagmus.

Whether or not this procedure will be valuable in research, I do not know. We found that a high percentage of patients with chronic sea sickness have an alpha wave rate of 10 per second above that of the normal population. The same observation has been recorded for patients with gastric ulcer.

According to Professor Neilson, mastoid inflammation has a high percentage of neurologic complications, as shown by the encephalograms. There are conflicting reports by others.

DISCUSSION OF PAPERS BY DR. BRUNNER AND DRS. SCHWAB AND CARTER

DR. JOSEPH E. J. KING, New York: A few years ago electroencephalography held only anatomic interest. Dr. Schwab's table shows that electroencephalographic studies gave the localization in 72 per cent of the cases, being outnumbered only by air studies. This is an astonishing record.

The cases of abscess are of especial interest. The electroencephalograms warranted definite localization of the lesion in 7 cases and probable localization in 7 others, and failed in only 1 instance. Three cases were listed in table 2 as subdural, and 1 as extradural. I have always considered a true abscess of the brain to be subcortical and should like to have Dr. Schwab clarify the terms. I should also like to have his opinion regarding the findings in cases of subdural collection of fluid, in special reference to the noninfected collection of clear xanthochromic or discolored fluid found in so-called serous meningitis and the massive collection of pus, which may not be walled off in the subdural space, in suppurative leptomeningitis.

My associate, Dr. Francis A. Echlin, with reference to a particular group under consideration, offers the following: In the acute phase of abscess, one finds a diffuse condition in the abnormality of the electroencephalogram, so that no lateralization can be made. If there is partial or complete encapsulation, the electroencephalographic findings become relatively normal except over the region of the abscess, where delta waves are prominent. Fluid in the subdural space, if enough is present, gives a flattening of the electrical activity. With marked increase of intracranial pressure, the picture is obscured; after dehydration the localization of the abscess most likely can be made. I should like to ask Dr. Schwab whether his experience bears out these statements.

Encephalography as described by Dr. Brunner is frequently carried out by members of the residence staff in the x-ray laboratory with which I am associated, without loss of time or transportation of the patient. We prefer the side position, and we use two small lumbar puncture needles. Roentgenograms of the skull are made during the substitution of air, which demonstrate the filling of the ventricle and give a clue to the amount of aid needed. As stated by Dr. Brunner, there is some danger, and the procedure should not be undertaken lightly.

I agree with Dr. Brunner regarding the contraindications. In the case of abscess with severe headache, a ventricular puncture is done preliminary to operation on the abscess. With infection there is always danger that encephalography will cause a rupture of protective adhesions.

Most otogenic and rhinogenic intracranial lesions demanding surgical treatment can be localized by proper clinical and electroencephalographic examinations. In doubtful cases, the making of a small burr hole through a small incision over the suspected lesion and the opening of the dura with or without puncture of the cerebrum offer advantages over encephalography. The condition can frequently be diagnosed, localized and adequately treated at the same time. If this fails ventriculograms can be made through the burr hole if air studies are indicated. In our clinic ventriculograms are found to be more accurate and satisfactory than encephalograms. The exception might occur in the case of unlocalizable abscess of the brain complicating osteomyelitis of the skull, or in cases of abscess of another type.

Air studies are preferable to puncturing the brain through an infected dura. When osteomyelitis involves the anterior portion of the skull, the posterior burr hole is preferred to the encephalogram. Should the osteomyelitis involve most of the calvarium encephalograms must be made.

The fact that ventriculograms require operative technic is not of sufficient weight in my opinion to warrant the choice of encephalograms. In any event the encephalographic procedure is not safe in inexperienced hands.

DR. FRANK R. SPENCER, Denver: I am indebted to Dr. Franklin G. Ebaugh and Dr. Charles E. Rymer for valuable information concerning the electroencephalographic test. They say it is easier to use for superficial lesions than for those

more deeply situated. This is important because of the enormous number of automobile accidents. If Drs. Schwab and Carter find it of value in 72 per cent of their cases, this is a high percentage of success.

The test may be of value in detecting malingerers who claim to be epileptic. It is of clinical value in cases of epilepsy, narcolepsy, tumor of the brain, subdural hematoma, abscess of the brain, meningitis and encephalitis; it is of value in cases of Sydenham's chorea for prognosis, in Schilder's disease for diagnosis and in cases of behavior disorder for diagnosis and prognosis.

In the Colorado Psychopathic Hospital 247 electroencephalograms have been made. An attempt has been made to localize tumors and identify post-traumatic lesions, subdural hemorrhages, encephalitis and the changes following prefrontal lobotomy. Normal alpha activity proceeds at the rate of 8 to 12 waves per second; beta, at from 18 to 36. There has been an attempt to identify the delta wave, which is slower than the alpha. In patients with epilepsy there is a slowing of the normal alpha wave. It is necessary to take tracings for a considerable time.

One patient with multiple actinomycotic abscesses of the brain was examined and I have the slide to show the electroencephalogram. So far we have no positive findings for abscess of the brain of otitic origin. Lesions of the cortex are detected more readily than lesions deeper in the brain. This test is not infallible and should be used in conjunction with all other known scientific tests.

Three slides were shown: (1) an electroencephalogram from a normal person; (2) one from a patient with status epilepticus; (3) one from a patient proved by autopsy made a few days after death to have had multiple actinomycotic abscesses. There were no actinomycotic abscesses in other parts of the body.

MR. HOWARD A. CARTER, Chicago: May I ask a few questions? What is the nature of the waves from the brain? Are they waves of electrical substances such as those found in a wire connected to a dry cell, or are they electrostatic waves or waves of a substance such as that found at one plate of the condenser? Does the bone of the skull offer resistance or is it dielectric, acting like a leaky condenser? What evidence have you that the weak electrical energies do not arise from the same causes as those energizing electrocardiographic activity? Has the activity of the sweat glands been eliminated? What assurance is there that the electrode has not been placed on a misplaced electrical motor point? Are the electrical energies actually coming from the brain or are they secondary in nature, involving the capillary bed?

DR. CHARLES T. PORTER, Boston: Dr. Schwab said something about the possibility of using this test in the prognosis of acute mastoiditis. Would it be useful in conditions simulating actual meningitis and other intracranial lesions?

DR. ALFRED LEWY, Chicago: Starting with the idea that electroencephalography might offer objective evidence of response to acoustic stimuli, I tested a totally deaf person, a person who was severely deaf and 4 persons whose hearing was normal. Prof. R. W. Gerard, physiologist of the University of Chicago, offered me the use of his laboratory, and the tests were run by Dr. Helen Carlson, who has done most of this work for Professor Gerard, Professor Kleitmann and others in Chicago. No constant findings appeared to indicate definite responses to acoustic stimuli. Tests were made in a darkened room with no sound but the hum of the motor present. The winking of the eye in nearly every case produced a wide excursion of the pen.

We found nothing to indicate that we could use this test for the detection of malingerers exhibiting deafness. If we could persuade the suspect to go to sleep so that we could get the waves characteristic for sleep, we might prove that an acoustic stimulus awakened him and in this indirect manner prove something.

Dr. Canfield told me he had tried this and I am sure that other members have had the same idea.

DR. MOSES H. LURIE, Boston: Since Dr. Seydell started the meeting with a historical reminiscence of the American Otological Society, I want to say that this

society in an indirect way was a great help in starting this work. Ten or eleven years ago, Dr. Gibbs and Dr. Bennett used the apparatus that we were using in the study of the Wever-Bray phenomenon to facilitate their work and almost stampeded Dr. Davis into electroencephalographic work. The members of this society feel proud in taking part in the development of this study of the brain.

Dr. Edmund P. Fowler, New York: I have tried to get some work done along this line in regard to malingering but have not succeeded. I should like to ask whether it would be possible in work done in regard to deeper localizations to determine any reaction in regard to tinnitus. Is there some electrical masking effect or change of phase which causes the change in these waves?

Dr. Robert A. Schwab, Boston: In answer to the question Dr. King asked with regard to subdural and extradural fluid, in 11 cases of subdural hydroma the electroencephalographic localization was not good. We located the subdural hydroma in 60 per cent of the cases but failed to obtain normal records in 30 per cent, and 10 per cent had absolutely normal records. The large subdural hydroma extending from the frontal to the occipital lobe, about 1 cm. thick, was removed by a neurologic surgeon. It had been associated with a normal tracing from each side. In other cases extradural fluid or pus has given us an electroencephalographic localization.

Sometimes the absence of the alpha waves in a given spot is useful in localization.

We have not tried hypertonic solution but have seen diffuse waves increased after suboccipital decompression for a ventricular tumor obstructing the aqueduct.

A patient with syphilitic arachnoiditis whose intracranial pressure was almost 800 mm. of water had a normal electroencephalogram on four occasions, also a normal ventricular wave. Postmortem examination showed nothing but arachnoiditis.

I am glad to learn from Dr. Spencer that they have the same apparatus available in Denver. The incidence of correct results throughout the country in proper cases runs about 75 per cent. In cases of lowered metabolism the alpha waves run down to 8 or sometimes 7 per second. They are increased by administration of thyroid, so the rate is probably a function of the cerebral metabolism.

This test is merely an additional tool, but as such it is good. In instances in which it is used as a supplement to other tests, especially air studies, if the tests agree the percentage of correct findings approaches 100.

In reply to Mr. Carter, I cannot define the waves in terms of molecular physics. Physicists have shown a difference in potential between the nucleus and the periphery of a nerve cell, which may be as high as 500 microvolts. When the nerve cell is discharged, this potential drops. It is like a little dry cell.

We have run these experiments on patients right through to death to see what happens to the waves from the brain. They disappeared some seven minutes before clinical death. After death, by pressing the chest the heart could be stimulated to a few beats, as shown on the electrocardiogram, but the waves from the brain were gone, so we know that in death the nerve cell loses its electrical potential.

We must be measuring the activity of these millions of brain cells. When the brain is damaged, these little pathways become detached from the centers in the brain.

Bone, skin, scalp and spinal fluid are all resistance. In cases of decompression, with the bone out, the resistance is less. It also varies according to the size of the electrode. The electrocardiogram has been known since 1917, but investigators do not know what all the wave forms are; these waves can be obtained from the heart outside the animal. The cardiac discharge has no relationship to that from the brain except as the vagus nerve controls the cardiac waves.

The waves are not from broadcasting stations to the skin, the blood vessels or the electrode itself, or the apparatus. These have been experimentally eliminated. We identify the myogram as coming from the muscle. We have obtained

these waves from the bare human brain. They appeared in the same characteristic manner but with a little higher voltage because of the lessened resistance. I cannot determine whether they come from the deep structures or from the cortex.

Dr. Lewy's observations on deafness agree with ours.

I can assure Dr. Lurie that Dr. Davis is running a large project for the United States Navy, on the one hand, and doing excellent work in the field of otology, on the other.

In answer to Dr. Fowler, we are unable to identify the tinnitus in the cases of Ménière's disease. We have done no experiments by placing the electrodes on the basal nuclei or under them. Presumably the waves are from the temporal lobe.

Dr. Hans Brunner, Chicago: I want to thank Dr. King for his comments. I stated that in cases of cerebral abscess with or without signs of pressure on the brain, encephalography is contraindicated. It might help if one could differentiate between communicating and obstructive hydrocephalus, but it is too dangerous. I also stated that lumbar encephalography should be done on strict indications and contraindications.

In cases of osteomyelitis it should be preferred to ventriculography. The neurosurgeon would certainly prefer ventriculography to encephalography, because it is easier.

One thing Dr. King mentioned was the danger of spreading meningitis. That is a form of chronic misunderstanding between the neurosurgeons and the otologists. I do not believe that today the otolaryngologist would neglect spinal puncture in well selected cases. I think the same applies to encephalography. It is up to the neurosurgeons to demonstrate that otologists break up adhesions as they claim.

The circumscribed labyrinthitis stays circumscribed even after rough work with the chisel. Occasionally it becomes diffuse labyrinthitis, which disappears in a week or two. Otologists do not destroy adhesions which are far more delicate than dural adhesions by chiseling just above the area. So how should they destroy adhesions by drawing off a few cubic centimeters of spinal fluid? This point should be demonstrated one way or another. Theoretic argument is insufficient as a reason for abandoning the otologic method.

Meningioma with Unusual Involvement of the Temporal, Sphenoid and Occipital Bones. Dr. Otto C. Risch, New York.

In a few cases an intracranial tumor has been reported both as meningioma and as endothelioma. The literature on the subject is quoted.

This type of growth occurs more frequently in females. Roentgenograms show principally (1) extensive destruction of bone without productive bone reaction and (2) preservation of the labyrinth.

The preservation of the function of all cranial nerves except the eighth was a striking observation. Pain in the temporomandibular joint is an important clinical symptom. Intracranial pressure did not appear to be increased. Cushing was impressed by the frequency of a history of trauma but did not accept the traumatic theory of causation in general.

In the present case the relation to trauma seems to be established as to time and location. Meningioma is most frequently located in the region of cell clusters of arachnoidal origin in the cranial base. Erdheim and Cushing were of the opinion that arachnoidal hernias were a consequence of an increase in intracranial pressure. Ruttin described such hernias in the petrous bones of rachitic children.

Woltman and Love presented a case of multiple meningioma with polyp in the right external auditory canal; the polyp proved to be a meningioma. The floor of the middle fossa resembled a sieve because of some small depressions into which projections of the dura extended.

When a bone is invaded, the tumor carries a bad prognosis because of the invasion of bone rather than because of malignancy. No type of therapy yields a cure.

In the case presented, the first symptom was pain in the region of the right ear following trauma. Two and one-half years later a polypoid mass presented in the canal. Examination revealed a dead labyrinth, rigidity of the neck, pain in the temporomandibular joint on motion and in the occiput on pressure. Roentgen study showed widespread destruction of bone with preservation of the labyrinth and no productive reaction of bone. At operation the mastoid cavity was filled with what was apparently granulation tissue. There was profuse bleeding. Histologic examination revealed meningioma. The original diagnosis of the polyp was papilloma and that of the operative specimen endothelioma. Reexamination of the tumor revealed its true nature. The patient is still alive.

DISCUSSION

DR. FREDERICK L. JACK, Boston: I was drafted for this discussion. It is a difficult assignment for one to undertake after a long time of passive retirement.

Many years ago I reported a case of hemangioma-endothelioma. Dr. Risch has thoroughly covered the subject of meningioma of the temporal bone. Early reports include one by Sir William Macewen and one by Dr. Keen of Philadelphia, who considered the finding to be fibroma.

I have a few observations on points suggested by Dr. Risch's paper: first, trauma as a cause. There is not sufficient evidence in support of this idea. One outstanding case was that of General Leonard Wood, a classmate and friend of mine, who was operated on by Dr. Harvey Cushing several times for a recurring psammomatous tumor. The initial injury caused a slight depressed fracture of the vertex. Growths of this type are potentially malignant. They were considered by Virchow and Paget as less malignant than carcinoma. Profuse bleeding after removal of a papillomatous growth suggests cancer. Dr. Crowe has stated that destruction of bone carries a bad prognosis. There was a dissenting opinion in Dr. Risch's case. Dr. Weller considered the case to be one of carcinoma.

I should like to ask Dr. Lisa if he found any psammomatous bodies in the tumor or the whorls characteristic of meningioma.

Cushing emphasized the importance of complete removal of all diseased bone and tissue. The number of cases in which treatment has been by operation in Boston hospitals is as follows: Dr. Munroe, the Boston City Hospital, 45 cases of meningioma since 1931; Dr. Horax, the Lahey Clinic, 60 cases since 1935, in about 19 per cent of which the growths were intracranial; Drs. Cushing and Eisenhardt, 2,000 cases, in 14 per cent of which the tumors were diagnosed as meningioma; the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary, 75 cases during the last ten years, which probably includes recurrences. The reports are confusing, owing partly to stenographic error but mostly to differences of nomenclature. The growths were not classified by Cushing as to location until 1922—cerebral convexities, anterior portion of frontal lobe, the region of the sphenoid and the sphenoidal wing.

Reports by Cushing and Crowe indicate that primary meningioma is almost never found on the temporal bone. Of Horax' 60 cases, 9 were orbitotemporal, arising in the lesser sphenoidal wing with a mortality of 16 per cent.

In my early years I reported a case of hemangioma-endothelioma, a "blood relative" of meningioma. I did not have the advantage of improved roentgen ray plates, of electrosurgery, of suction, of control of hemorrhage by muscle or of blood donor. Cushing stated that brilliant achievement of one day becomes commonplace the next. The finding of masses of granulation tissue with unusual hemorrhage in Dr. Risch's case suggests the gross appearance of hemangioma-endothelioma, but the barrage of eminent opinion on his side gives me reason to pause.

DR. JAMES R. LISA, New York: There is general agreement that meningioma is arachnoidal in nature and that the type cell is the meningocyte. As to whether

it is ectodermal or mesenchymal is in dispute. In some cases it shows a lack of reticulin formation and appears to be almost purely cellular. If it is of mesenchymal origin, one might expect reticulin formation to be prominent. When this formation is found it may be merely a fibroblastic response to the tumorous cells.

The cases of meningioma similar to that reported by Dr. Risch have been the ones in which the meningioma has been most frequently mistaken for endothelioma. The terms endothelium and endothelioma have been used with different meanings. Maximow believed the term should be limited to the lining of the blood vessels, the lymph vessels and the heart. The term endothelioma has been used loosely. Ewing has expressed the belief that the separation of meningioma from endothelioma is academic only. The endothelioma, however, has one distinguishing feature—its ability to form reticulin.

In the study of neoplasms and the potentialities of the type cells one's observations must be meticulous and one should be careful in the use of terms.

Dr. Jack asked whether I found some psammomatous bodies or whorls characteristic of meningioma. In the present case, the psammomatous bodies were not present, and the presence of whorls was doubtful. I do not lay much stress on this point since the same appearance is present frequently in other tumors not meningiomatous in nature and anaplastic in type.

Impaired Hearing in School Children. Dr. Samuel J. Crowe, Stacy Guild, Ph.D., Dr. Ella Langer, Dr. Walter Loch and Mary H. Robbins, M.B., B.S., Baltimore.

Our report two years ago on 1,365 white children in the schools of Baltimore showed that the most common defect of hearing in children is impairment of the thresholds for some or all of the frequencies higher than 2048 cycles per second. Nearly 40 per cent had this defect. Only 3 per cent had impaired hearing for all tones or for the spoken voice.

The report also showed that regrowth of lymphoid tissue occurred in over half of the children who had undergone adenoidectomy. A majority of these children had lymphoid tissue in the fossa of Rosenmüller and in the region of the orifice of each eustachian tube; 208 children with hypertrophied lymphoid tissue and impaired hearing were improved by irradiation of the nasopharynx; 152 have been followed up over a period of two years.

The conceptions underlying the treatment may be summarized as follows: When the ventilation of the middle ear is interfered with, the differences in pressure cause retractions of the tympanic membrane and impairment of hearing, and the result is mucosal hyperemia and edema. Hyperplasia and fibrosis may develop. We find that the perception of high rather than that of low tones is impaired first. Probably this was not observed before the advent of good audiometers because the routine threshold determination for high frequencies was 2048 vibrations. Loss for high tones was not checked by tuning fork or voice.

A control group was selected for the evaluation of the role of spontaneous regression. Thirty-one per cent of the controls showed spontaneous regression of lymphoid tissue to the extent that both tubal orifices were normal. In 15 per cent there was regression but not to the same extent. Of the 152 children whose treatment was started in the year 1939-1940, 136, or 89.5 per cent, showed the tubal orifices normal and the adenoids small or absent. The average number of treatments was less than four; only a few had more than six. One boy had had nine treatments without success. It is our opinion that regeneration of lymphoid tissue occurs less often after irradiation than it does after surgical removal of the adenoids.

None of the children suffered burns or complained of sore throat after treatment. A few adults similarly treated have complained of pain and have experienced exacerbations of chronic nasopharyngitis. All children who have had otitis media since treatment was started had histories of recurrent otitis. Most children with histories of recurrent otitis have had no attacks since the lymphoid

tissue was reduced. The balance is unquestionably in favor of the use of radon as a therapeutic measure. It should not be used during or soon after an infection of the upper respiratory tract.

The value of therapy for hearing defects can be determined better by other than statistical observations. The average of the changes for low tones is from 1.5 to 4 decibels of improvement; for the middle range—512 to 2896—the average change in two years was less than 1 decibel; for the high frequencies—4096 to 16,384—the average impairment at the last test was greater than that at the first. The best explanation seems to be that these children were two years older at the last test and that various lesions that cause impairment of hearing for high tones had had more opportunity to exert their effects.

A special study of seasonal fluctuations will be published elsewhere by Miss Robbins, as well as a study of "tonal dips" by Dr. Loch.

In the first group who had impaired hearing for high tones only, the boys outnumbered the girls 3 to 1. The same ratio was noted in the other groups. It is recognized that some of these had lesions other than those secondary to tubal occlusion. Nevertheless we treated the nasopharynx in order to decrease the danger of a progressive conductive lesion being superimposed on the cochlear lesion if this were present. We believe it is important to do everything possible to prevent development of a lesion of the middle ear in children with an impairment of hearing due to a defect of the inner ear.

Acuity for high tones improved in one third of the ears with hearing defects limited to this range. Sometimes improvement began soon; sometimes, not for several months. Tubal inflations have not been used; perhaps they would have hastened improvement in some. The results warrant the conclusion that impairment for high tones was caused by lesions of the middle ear that regressed after treatment, and perhaps further deterioration from lesions of the middle ear has been prevented.

The impairment was essentially the same at the last as at the first test in slightly less than one third of the ears with impairment for high tones only. The diagnosis must remain uncertain. Among the children who had poor hearing for other than high tones, the type of impairment was a loss of 25 to 40 decibels for low and middle tones but somewhat more for high tones.

With impairment greater than this in the better ear, the child seldom can stay in regular classes. Many of these children had improvement of 15 to 30 decibels except for the high tones after radon therapy and were removed from the special problem classes. Those who have not improved either have chronic suppuration or have had repeated attacks of otitis. They have sustained permanent damage to the structures of the middle ear.

Handicap through deafness could be prevented in many cases by prompt medical care of any patient with acute otitis media. Lay organizations can materially help by stressing the importance of proper medical care of all children with acute otitis media. The possibility of success in such a campaign is not limited to large communities with special equipment and personnel. More deafness can be prevented during the next ten years by nonspecialists than will be cured by the personal activities of all the otologists. Too often the deafened person goes to the otologist when it is too late.

Children with impairment for middle tones of more than 40 decibels were not in the regular schools and did not come to our clinic. For those who have excessive amounts of nasopharyngeal lymphoid tissue prophylactic treatment is advised to prevent progressive lesions of the middle ear. The condition of the teeth, the weight, the height and other anthropometric observations disclosed nothing that affected the changes in hearing.

The practical conclusions that may be drawn follow:

1. Acute otitis media must be fought in all cases. To this end, educational campaigns must be organized that will give physicians the chance to treat promptly all patients with otitis media.

2. Radon therapy is a useful addition to methods for reducing hyperplastic lymphoid masses. Approximately 50 mg. of radon can be rented for $15 a month. If only 15 to 20 patients a month are treated, the cost of the radon is the smallest item in the expense.

3. It is possible to restore hearing in many children with impairment of hearing for voice and thus simplify the educational problem. An audiometer is not necessary to detect losses of hearing in children. Any one with common sense, a watch and a voice can make the test. Loss of conduction by bone has unreliable prognostic value. The decision to treat should be based on a combination of impaired hearing and hyperplastic lymphoid tissue in the region of the eustachian tube. If an audiometer is available, it should of course be used.

4. Parents whose children do not improve after treatment should be instructed in what modern hearing aids can do. In many communities it would be necessary to educate the educators. As to children with loss for high tones only, we believe it would be a mistake to try to find them until preparations are made to do something for them.

If a good audiometer is available and if there is a budget for a properly trained examiner and a good otologist who is willing to make physical examinations, treat the patients and reexamine them periodically for years; then, and only then, is it practical to start the search for children with hearing losses for high tones only. This aspect of preventive medicine is not spectacular.

DISCUSSION

DR. HORACE NEWHART, Minneapolis: This paper is based on an ideally thorough research project. It gives to the subject of the conservation of hearing the recognition which this important problem deserves.

Notwithstanding improvement in the technic for early discovery of hearing deficiencies, progress has been disappointingly slow for many reasons. First, there has been no adequate instruction in the prevention, treatment and amelioration of impairment of hearing. Instruments for mass testing of school children yield fantastically different results. It is recommended that only acceptable audiometers be used in carrying out the school health program and that the operation of these instruments be restricted to properly trained nurses and technicians. In Minneapolis, of 12,000 children from the fifth to the ninth grade 3.2 per cent were discovered by the older methods to have hearing deficiencies worthy of further scrutiny. With a pure tone audiometer, 19.8 per cent of 6,300 pupils were found deficient, many because of loss of hearing for high tones. This loss occurred in 14.4 per cent.

Here is a slide shown with the permission of Dr. Bunch. The loss of high tone was not detected by the phonograph audiometer. There is a deplorable need for authentically standardized, uniform criteria.

Wide discrepancies in the incidence of hearing defects reported from different communities have created a lack of confidence in the methods used. There is urgent need for a program of medical follow-up and additional adjustment for those children found to have defective hearing. A wisely planned campaign will create a sufficient demand for legislation to provide the necessary funds for a satisfactory program. The active leadership of members of the medical profession is needed for the success of such a program. This paper affords a basis for such a campaign.

As a simple masking device, I wish to show the following: a small fruit juice glass, absorbent cotton placed within it, on top of which is attached a telephone cushion of sponge rubber. This at least keeps out some of the prevailing noise about one.

DR. EDMUND P. FOWLER, New York: Otologists should keep in mind the pitfalls in all this type of work. They are using more than just one treatment. Instructing the parents as to the general care of these children and all that goes with it is part of the program.

The eustachian tube has been referred to as the U tube by several of my patients—not a bad abbreviation. It is difficult to tell whether or not lymphoid tissue is obstructing the orifice. A child with perfect hearing may have masses of lymphoid tissue around the tube. A child in whom the tissue is far from the tube may have marked deficiencies of hearing. A drop, less than a minim (0.06 cc.), of mucus will close the tube until the air bubbles through. It is difficult to diagnose the patency of a eustachian tube. The surest way is to look at the drum after the patient has had inflation or other manipulation to see the drum move.

I have irradiated many children with the method Dr. Guild and Dr. Crowe are using, mainly in the cases in which the impairment is more severe. I have not been satisfied with the shrinkage of the lymphoid masses but have seen the mucus diminish. That may be one of its great efficacies.

Loss for tones of more than 4000 double vibrations does not amount to much in the interpretation of speech. In testing for hearing of high tones, a sound-proof or quiet room is necessary, and the child must be retested several times because one gets standing waves with these high tones, which have marked effects as far as the decibel response is concerned.

Lymphoid tissue in children tends to recede at puberty. After its surgical removal, nature tends to put it back, probably from some protective impulse. This does not occur when it is gradually shrunk by irradiation. If one has a series of 30 patients and 25 show slight loss and the other 5 recover or show tremendous improvement, it brings the average up greatly and yet only a few have shown improvement.

I think the recruitment test is the only way in which one can determine whether high tone losses are due to lesions of the auditory nerve or to lesions of the conductive apparatus or to both.

For the past two years my associates and I have examined 150 children suffering from severe losses, with controls, as to their intelligence quotients and psychic makeup and hope to have something to present in another year or so. We have already seen many show remarkable improvement who have had no treatment whatsoever. Perhaps if we used the one ear on the same child as a control we should have more accurate data. We have not yet definitely decided what constitutes a real loss. As physicians we are interested in the therapeutic approach, but the school authorities are interested in the educational problem. The child with no more than a 30 decibel average loss is not handicapped if he sits near the front of the room.

Dr. Burt R. Shurly, Detroit: In the school system in Detroit 125,000 children were examined with phonographs, and an average of 4.3 per cent were found to have some loss of hearing.

One is dealing with an educational problem—in the first place the problem of the school teacher and in the second place the problem of the parents.

Some one in the medical profession read an article on the large loss of life due to poliomyelitis following tonsillectomy and immediately the Board of Health of Detroit stopped operations for the removal of tonsils among the school children. This movement is the other way from the excessive operative notion which physicians once had. A tonsillectomy is more beneficial than anything the otologist does if it is done properly. This era of changing and hysterical ideas is bad for members of the medical profession. The children in Detroit are followed up through a card referring them to the family doctor, and a school nurse follows that through. A reexamination is made in three months and another if necessary.

I believe the operation for removal of adenoids when properly indicated will be of more value when the younger medical graduates learn to do it carefully and thoroughly. A few cases of poliomyelitis in a community should not scare the board of health and the parents.

Stacy R. Guild (Ph.D.), Baltimore: The time has come to do something for these children. I am delighted that so many people are interested in doing something about it.

Fenestration of the Labyrinth: A Report and an Analysis of Cases in Which the Operation Was Performed. DR. H. EDWARD CAMPBELL, Philadelphia.

A careful analysis will show that the actual results lie somewhere between the pessimistic point of view of the one group of surgeons and the overly enthusiastic one of the other group of operators. Years ago I reported results in a series of 30 cases. I shall make a follow-up report on these first and report, in addition, 23 cases in which fenestration was done eight months or longer ago. The last three preoperative audiograms were averaged.

Follow-up Report on the First Thirty Cases.—Twenty-one patients had proper indications for this operation; in 13 hearing improved 14 decibels or more, the average improvement being 23.6 decibels. One year ago the average for these same patients was 21.8 decibels. Eight patients in whom fenestration proved unsuccessful show an average loss of 3 decibels, compared with an average loss of 4.8 decibels a year ago. Four patients in whom the fenestrations were revised show no change. The fistula reactions of the 13 with improved hearing remain the same. There is no increase in tinnitus in the cases in which the operation was successful. There have been no complications in any of the cases. Of the ears that were not operated on, some show a slight loss and others a slight gain; there has been no gain or loss of more than 10 decibels in any one case.

Results in Twenty-Three Additional Cases.—Twenty-two patients had proper operative indications. Eight had apparently normal tympanic membranes and true otosclerosis; 10 apparently had so-called catarrhal deafness; 4 showed scars in the tympanic membrane, and 1 had middle ear deafness. Twelve patients were operated on according to the Lempert technic. Eleven were operated on by a combined postauricular and endaural incision.

Thirten patients received an improvement of more than 12 decibels, the average being 21 decibels. However, the remaining 9 experienced either a negligible improvement or a loss. The greatest improvement was 36 decibels after one year. The 9 whose fenestrations proved unsuccessful had an average loss of 6.7 decibels, but 4 of these had a 2.9 decibel improvement. Of 8 patients with normal tympanic membranes, 5 showed a substantial gain; of 10 with thickened membranes, 7 had a gain but not as great a gain as the otosclerotic group. Of 4 patients with scars, 1 lost 12 decibels while 3 showed gains of 24, 12.7 and 6.7 decibels. Of 10 patients with profound deafness, 6 noted a substantial and 1 a slight gain, while 3 lost. In all but 1 of the 9 whose result was unsatisfactory the loss occurred during the healing process. One fenestration was revised without success. Among the 13 patients with improved hearing the fistula reaction was 3 plus in 2, 2 plus in 7 and 1 plus in 4. Of the 9 patients whose fenestrations were unsuccessful, only 2 retained a mild fistula reaction. In all of the patients whose fenestrations were successful the tinnitus was reduced in proportion to the improvement. In the unsuccessful cases the failure was attributed to closure or partial closure of the fistula.

In the 23 cases the errors of technic were as follows: (*a*) In 2 cases the tympanomeatal flap was lacerated. Remnants of the flap were placed over the fistula. The end result was 36 and 24 decibels of improvement. (*b*) In 1 case the tympanic membrane was perforated but the laceration healed. (*c*) In 1 instance the incus was dislocated and removed. The improvement in hearing was negligible.

The postoperative complications were as follows: (*a*) facial paralysis occurred in 1 case. Function was restored in four weeks. (*b*) Postoperative infection developed in 5 and was controlled by sulfathiazole. There were no bad effects. (*c*) In 2 cases the external auditory canal became constricted. (*d*) Slow healing was observed in 2 cases.

An attempt has been made to present facts in a fair and unprejudiced manner. The extremely critical analyst will find considerable material for condemnation; the enthusiastic optimist will discover sources of great encouragement in the

report of the results, particularly in the end results in selected cases. It is hoped that the majority will find the results sufficiently favorable to justify continuance of this work as a therapeutic procedure, even though they realize that the results so far obtained are far from perfect. The necessity for further research to advance such a promising start is obvious.

DISCUSSION

DR. NORTON CANFIELD, New Haven, Conn.: Dr. Campbell's two reports represent the best clinical analysis which has thus far been presented. I am sure no one appreciates more than Dr. Campbell does the difficulty involved and the details which should be improved.

All men of experience know that impaired hearing is a variable phenomenon and is often influenced favorably without operative procedure. No statement has been made as to the preoperative care in any of the cases reported, nor any attempt made to evaluate the possibility of nonsurgical treatment before operation, with minor exceptions.

I have been following some cases for a period of two or three years, in which improvement without operation has been striking. I wonder how many in Dr. Campbell's series of patients with favorable results could have obtained such results without operation. Case 13 in the 1941 report may be one illustrating this point (three progressive audiograms show an improvement of 14, 33 and 31 decibels over a period of one to three years). Many cases of deafness are not hopeless, for hearing may improve under favorable circumstances.

Dr. Campbell's method of demonstrating improvement by averaging the decibel change at 512, 1024, 2048 cycles may have some clinical advantages, but in many cases of deafness an improvement of 20 or 30 decibels at 2048 cycles may markedly improve interpretation of speech, whereas the same improvement at 512 cycles may not lead to an equal improvement in the interpretation of speech although the percentage of improvement for this calculation may be the same. From Dr. Campbell's figures, 7 of 12 patients with otosclerosis, or 58 per cent, improved significantly; 14 of 20 patients of the catarrhal group, or 70 per cent, improved and 6 of 11 who had had suppurative otitis media, or 54 per cent, are all included in the number with successful results.

One illuminating comment in both reports seems to be conclusive evidence that clinical improvement in hearing does not depend on a precise operating technic. Infection has apparently not interfered with results. The procedure is evidently still one of unknown factors. I have tried to establish these factors but have been unsuccessful. I again plead for a research program in which this and other problems of hearing would be investigated. Dr. Campbell's paper should encourage otologists to spend more time on the fundamental elements of hearing rather than on minutiae of surgical technics, which have been shown to be unimportant. I consider Dr. Campbell's report of cases the best clinical presentation since Holmgren's in 1938.

Certain questions in connection with fenestration need to be answered:

1. Was tinnitus worse after the unsuccessful operations, and if so has any treatment been effective?

2. What pathologic change is responsible for the deafness in the "catarrhal cases"?

3. What change takes place in the vascular supply of the inner ear incident to the operation?

DR. D. E. S. WISHART, Toronto, Canada: It is a pleasure to criticize a paper which rings so true. Success in 59.9 per cent of Dr. Campbell's cases is equivalent to admitting some of the claims of enthusiastic supporters of this operation. The opposite side of the picture is the closure of the fistula in 40 per cent.

Dr. Campbell has shown that success and failure are not necessarily due to perfect or imperfect surgical technic, respectively. An important fact emerges,

i. e., that the secret of how to keep the window open is not yet known. If it were known, a certain surgeon of genius would not have been forced to shift to the fenestra novovalis.

Dr. Campbell's new figures support his previous conclusion that patients who have retained their hearing improvement more than eight months will retain it permanently and that the greatest benefit is achieved at the moment of opening the labyrinth; i. e., the cure is mechanical because the cause is mechanical.

One case otologists must all think about—that in which the greatest improvement, 36 decibels, occurred, which was not reached until twelve months after fenestration. What factors caused the postoperative course to be so different from the average?

Dr. Campbell concludes that it is not yet known whether or not the operation will prevent the progress of deafness in otosclerotic patients. The final success in his series will not be known for five years. At that time the members will want to know how many of the patients have been able to gain or retain employment and carry on without a hearing aid. That, in my opinion, is the real question in regard to this operation. Otologists know that in otosclerosis deafness may advance or recede or remain stationary and that histologic otosclerotic changes have been found when there was no clinical deafness. Some factor must govern this variation. This paper affords ground for belief that the relief of deafness from otosclerosis in its earlier stages will depend on the otologist who is a good physician rather than on one with surgical skill.

Dr. Robert L. Moorhead, Brooklyn: My remarks are based on 17 cases. One patient, operated on thirteen months ago, has splendid hearing, but the majority have been under observation too short a time and will be reported on in detail next year.

Dr. Campbell's analysis includes his bad as well as his good results and an attempt to find the cause for the failures. This is characteristic of the good surgeon. In none of the cases in Dr. Campbell's series has a diminution of hearing been shown after eight months. I think one may narrow one's findings to the frequencies 512, 1024 and 2048 for practical hearing. A gain from a 70 to a 50 decibel level of loss is of no practical use to the patient. We should report results as good only when the hearing has been restored to a 30 or 33 decibel loss, or better. This will give a more satisfactory picture of practical results, which is the only basis on which the success of the operation should be judged. I do not believe the operation is indicated unless the hearing has diminished to a 45 decibel level or lower.

In Dr. Campbell's last series he reported only 1 case in which proper operative indications were not present, in contrast to 9 in the first series. Eliminating unsuitable cases will go a long way toward improving results. In 11 cases of the second series the operation was performed by the postauricular route. I fail to see any advantage in this and believe it has definite disadvantages, adding to the reaction as well as to the danger of infection. The placing of the new window in the vestibule is also more desirable than fenestration of the horizontal canal. If Dr. Campbell will try this, he will find there is no initial rise followed by a loss, but a steady improvement commencing two or three weeks after operation.

I regret the emphasis on good results in spite of technical errors. I fear this may encourage more to undertake the work without adequate preparation. Only by meticulous care will the best results be accomplished.

Stacy R. Guild (Ph.D.), Baltimore: I am going to project some lantern slides which Dr. Campbell, on his visit to Baltimore, asked me to demonstrate.

The inner structures and relations are normal. They show that to do a fenestration in the vestibule above the oval window it would be necessary to sacrifice either the facial nerve or the superior division of the vestibular nerve.

The last section shows why with a low tegmen typmani it is almost impossible to expose the region satisfactorily by either the endauricular or the postauricular approach.

Dr. Jacob L. Maybaum, New York: I have watched Dr. Lempert operate, I should say, seventy-five times. I have done much work on cadavers and have operated in 12 cases with increasing good results. Nothing can be gained by belittling such work or by indulging in extravagant claims for it. I wish to report an improvement dating back four years in a patient I had seen repeatedly over a period of seven years with undoubted otosclerosis, who was wearing a hearing aid. After the operation he heard a whisper at 6 feet (183 cm.) in each ear. At the time of operation he heard a tone of 256 cycles per second at 60 decibels' loss; in a recent test, at 30; he heard a tone of 512 cycles at 55 then; now, at 30; a tone of 1024 cycles at 45 then; now, at 25; a tone of 2048 cycles at 45 then; now, at 20. He has discontinued use of the hearing aid and is now entirely rehabilitated as a result of the fenestration done four years ago.

I have followed 4 other cases in which the operation was done a year or more ago, with similar results.

I think Dr. Campbell should have stressed the fact that although he obtained good results in spite of certain operative accidents, those accidents do not represent a method which will bring about telling results in such work. He will probably say, as all who are doing this work know, that the best results will be obtained by close adherence to Dr. Lempert's technic.

I am encouraged by the results in patients operated on three, three and one-half and four years ago whom I have tested in my office repeatedly over a period of years. I believe continuances of this work by more otologists who will make every endeavor to acquire a technic approaching that of Dr. Lempert before they operate will give good results.

I do not believe the entire problem of otosclerosis is solved by this operation.

Dr. Frederick L. Jack, Boston: In 1892 or 1893 members of this society held meetings on the subject of the improvement of hearing by operation—stapedectomy. I wish to say a word because I think Campbell and Lempert have something. The idea then was to get a sound wave into the receiving apparatus, skipping the conductive mechanism, which was out of order.

My colleagues and I worked on that idea for a while, then there was a hiatus for some years and nothing was done until the recent resurrection of the idea. Are the results permanent? In Lempert's case the results are still good after four years. He is getting out of the woods pretty thoroughly, but other things may come on eventually. Another question which has not been answered is: What effect does the operation have on the initial disease which caused the deafness? The disease is going on and may finally conquer, and where will the otologic surgeon be? I think something useful is coming out of this, and I shall be glad to see it, because I have followed it for fifty years.

Dr. H. Edward Campbell, Philadelphia: I will first attempt to answer Dr. Canfield's questions. Yes, the tinnitus has been worse after unsuccessful operations, i. e., in patients who have lost their early improvement. In general, the decrease in tinnitus has been parallel with the amount of improvement.

The second question: Is a pathologic change responsible for the deafness in catarrhal ears? I found it hard to make an accurate diagnosis of otosclerosis, going largely on the absence of a history of pathologic processes in the ear, a normal tympanic membrane and a patent eustachian tube. A diagnosis was made of probable otosclerosis with conductive deafness. Those conditions in which there are obstructive changes in the drum membrane, I classify as catarrhal. The loss of hearing in those cases I suppose to be due to fibrosis. I do not know exactly what those processes are. The patients usually gave no history of suppurative otitis.

As to the vascular supply in the inner ear subsequent to operation, I can make but a vague answer. The interruption or change of the blood supply in the region around the capsule might lessen the deposition of bone in cases of otosclerosis. It is merely an idea.

Dr. Canfield's point regarding preoperative treatment is good. Many of my patients have worn hearing aids and received no improvement by ordinary methods of treatment. In future, I shall go into further detail as to what preoperative treatment has been done.

I did not mean to emphasize that slipshod technic might have a good chance for improvement. I mentioned that point to show that one knows little of the factors concerned in the preservation of the fistula.

Dr. Guild's slides show something important—just what tissue is sacrificed in the fenestration operation. I have done fenestration of the vestibule in 10 cases and find a distinct improvement over the older method, but the cases are too recent for an opinion.

Whether or not the superior vestibular nerve is sacrificed, as Dr. Guild remarked it may be, I do not know. Perhaps he can tell what effect that would have on vestibular function.

The Case for the Hearing Aid. DR. GORDON BERRY, Worcester, Mass.

What can be expected from fenestration and what help may be secured from a properly fitted hearing aid? I have not performed that operation, and I do wear a hearing aid to which I am partial. This does not prevent me from seeking and reporting evidence presented by the leading exponents of the operation and contrasting it with claims of hearing aid manufacturers. The literature concerning the operation consists of the successive reports of a few leaders, together with reports from less experienced operators, who offer comparison of their work with that of the leaders.

The early reports presented many failures, but otologists are seeking to discover what a skilled operator can now hope from fenestration. I have selected the reports of Shambaugh Jr., Fowler Jr., Lempert and Campbell. In 1938 Shambaugh reported his first 12 cases and found the results discouraging. Since then he has reported a total of 72 cases, 15 with revisions, and of the total number of patients 60, or 83 per cent, obtained satisfactory improvement. Each one of these who had used a hearing aid before the operation has better hearing than was obtained from the hearing aid. Then he (Shambaugh Jr.) added that "the operation is still in its infancy, and few otologists are qualified to carry out the . . . operation." Another article by him reported the results of 128 consecutive operations on 117 patients up to December 1941; 65 of these patients enjoyed an apparently permanent average gain of 25.7 decibels for the speech frequencies—a splendid showing. He did not show his failures.

Fowler Jr. was not so optimistic in view of his experimental work on monkeys, checked by every detailed scientific means. He made the following points: Careful anatomic measurements show that the cutaneous flap cannot be made long enough to cover the fenestration. When Thiersch grafts were used on monkeys, they tended to form epidermal inclusion cysts. The usual end result was bony closure of the labyrinthine fistula.

Hughson's work on round window grafts gave as his final average maximum gain 16.25 decibels. His routine gain ranged between 5 and 15 decibels at some frequencies and questionable gains at others.

Lempert reported the largest number of fenestrations. He listed 375 cases of his series. In 300 of these the window was made in the external semicircular canal, and in 75 it was made in the vestibular dome. In these 75 cases, he claimed, there had been no bony closure. The audiometric results in 18 selected cases are given. I have worked out the averages for both groups. In the first group, 10 cases, the average gain for seven frequencies was 18 decibels. In the second group, 8 cases, the average gain was 20.3 decibels. The favorable over-all average gain for these 18 selected cases was 19 decibels. For frequencies 512, 1024 and 2048 the first group showed a 27.5 decibel gain; the second, a 24.3 decibel gain. These are good gains, but they were obtained in selected cases and probably are maximum gains.

Campbell insisted that the endosteum should not be removed and that Shrapnell's membrane rarely reaches far enough to cover the window; he prefers that the under surface should not be lined with skin. Thus two leading operators differ on essential points. This may indicate confusion among the advocates of the operation. In the 13 selected cases reported by Campbell with an operative average gain of 21.8 decibels, the best single gain was 35.7 decibels, but the patient who had the worst result suffered a loss of 30 decibels. The average of the entire series of 30 cases was a slight over-all loss but a gain for the speech range of 3.7 decibels. His more recent cases have shown an average gain in the conversational zone of 7 decibels. This shows a difference in results between a selected group of cases and those of a consecutive series.

Now I shall show what a well fitted hearing aid can do. Mr. Watson, of the Maico Company, Mr. Huth, of Aurex, Mr. Howard Carter of the Council on Physical Therapy of the American Medical Association, and Mr. Kelly, of the Bell Telephone Laboratories, have furnished selected figures.

Mr. Watson furnishes thirteen graphs showing gains as follows: (1) 40 decibels at frequencies 1024 and 2096; 30 decibels at 512 and 4096; (2) 50 decibels at 1024 to 4096; 30 decibels at 512 and 5792; (3) an average of 42 decibels for the entire range; (4) 50 decibels for the entire range; (5) useful hearing in a patient with a 90 decibel loss; (6) useful hearing in a patient with a sudden localized drop in the 2000 to 5000 range; (7) 25 decibels at 512 and 1024 when only a carbon aid was used but 60 decibels when the patient was fitted with a vacuum tube aid. One patient showed a marked gain with a binaural fitting.

The acoustic engineers also recorded performance by "pressure calibrations." This amplification yields too much noise in the lower frequencies but the tone discriminator can cut down the troublesome low tone magnification.

Mr. Huth's chart shows that the vacuum tube instrument can furnish a constant amplification of 60 decibels between 300 and 4000 cycles and 50 decibels between 200 and 5000 cycles. Milder forms of deafness do not need these extreme amplifications.

Carter's material in the report of the Council on Physical Therapy in *The Journal of the American Medical Association* describes the external characteristics of twenty accepted instruments—battery consumption, response to articulation tests, amplification at threshold and 45 decibels above. The vacuum tube types begin to help only around 500 cycles. Frequencies of 1024 and 2048 offer the most gain, decreasing gradually at 4096 and sharply above that frequency. For the conversational range the average amplification of the entire group is 30 decibels, officially listed performances reported by the Council on Physical Therapy.

A comparison may be made of 2 cases (chart 16 in the original manuscript) of similar deafness; the hearing graphs in these cases almost ran together in the low frequencies, the case in which fenestration was done showed 20 decibels better at 2048 cycles, but the correction shows a 20 decibel advantage for the case in which a hearing aid was used.

Perhaps a fairer way is through over-all averages. The only over-all average I have is the questionable gain enjoyed in Campbell's entire series, which would contrast unfavorably with any demonstrated gains from modern well fitted hearing aids.

With the exception of the reports of Hughson, Shambaugh Jr. and Lempert, I have found no carefully prepared article showing considerable and consistent gains from operative interference. The average for Lempert's eight selected fenestra novovalis operations gives an over-all gain of 23.3 decibels and a gain of 24.3 decibels in the conversational range. A composite picture of Mr. Watson's 13 cases shows an over-all average gain of 43 decibels. The Watson series starts with an average of 12.5 decibels less hearing before the hearing aid was used and a more profound degree of deafness amenable to help. Also, please remember that the surgeons' failures are many, while the failures with hearing aids are few.

There is one more method for estimating the efficiency of a hearing aid. The otologist can measure its performance in a quiet environment by testing the

patient's capacity for conversation with and without the aid. He can also use the West syllable test. It seems inescapable that a well fitting hearing aid offers more hearing at less expense, less hazard and less mental worry. Why operate?

There are several valid reasons for operating. 1. There is the challenge to the otologist. He wishes to compare well with best. 2. There is the gamble that any patient may enjoy a 35 decibel gain. 3. The patient does not wish to advertise his infirmity; he will go through any sacrifice to escape wearing a hearing aid. Common sense and kindly guidance have persuaded people to wear glasses and false teeth. 4. Repair of and replenishment of instruments are a nuisance and expensive, but the regular trips tc the operator are more taxing and who knows whether or not the bony membrane will close? The claim that early discovery and prompt operation stop the progress of deafness is important but has not been proved true. Thirteen of the patients Campbell has operated on have shown a gain of 1.8 decibels during the past years.

I would urge those who have operated to check back through their cases. There are two suggestions to be derived from this review: 1. He who recommends an operation must in fairness to his patient inform him more accurately on the performance of a well fitted hearing aid. The instruments are becoming better every year. 2. The acoustical engineer must devise a more accurate method of fitting hearing aids. The average salesman is not particularly well informed on acoustics. Fitting should be done by an acoustical expert.

This discussion prompts an admiring tribute to the skill of the surgeon striving to help the hard of hearing patient. Hearing aids show an average improvement of 43 decibels, against 23 decibels for the operation. In reports of results from the operation, the bad should be given with the good, in a consecutive series. Only Campbell's results permit a fair appraisement. A similar series should be reported by acoustical engineers. We do have the Council's report showing an average gain of 30 decibels for 18 hearing aids listed.

Further study of the important claim that fenestration stops the progress of advanced deafness is necessary. The patients with the less severe deafness, best suited for operation, are just the ones most easily helped by hearing aids. A well fitted modern hearing aid gives greater amplification than an operation, to a wider group of frequencies and, in spite of the nuisance factor, at less cost, less pain and less worry. Greater efforts are urged on the part of acoustical engineers.

Dr. Gordon Berry, Worcester, Mass.: I present the reverse side of the picture in behalf of hearing aids. All will agree that Dr. Campbell presented his material well, and I confess to a prejudice in favor of hearing aids, but I am trying to be fair in the assembly of my material and its presentation.

DISCUSSION

C. C. Bunch (Ph.D.), St. Louis: Patients with a hearing loss of 30 decibels or less frequently come for help. They usually derive no benefit from a hearing aid—they hear too much noise in the instrument. They may use one but only under special conditions. Fenestration to be successful should show that it makes the hearing better up to a 25 or 30 unit level. Dr. Campbell mentioned it, I think, for the first time.

The second point is that audiograms were shown, some of which were made when the patient had no hearing aid on and others when the patient had one on. I have not been able to secure that type of hearing test. I tested a patient with a 70 unit loss, using a telephone receiver. She was wearing a vacuum tube hearing aid. I placed sponge rubber between the microphone and the receiver of the hearing aid and used a heavy weight to keep these as closely coupled as possible. At 1024 cycles I was unable to find a threshold of hearing. The hearing aid was picking up sound from some place, which did not come from the audiometer. I could do it with a phonograph but not with the phonograph audiometer, which made a clear tone. Furthermore, the audiometer was making a howl. I investigated this, even disconnecting the batteries, and found I could pick up the sound

without hooking up the latter. It was impossible for me to determine by using an audiometer how much better a person hears with his hearing aid. I wonder how those charts are worked out. To my mind, conversation is the best test.

I am not capable of speaking on the technic of the surgical procedure. If this operation is successful, people who have had it will not seek a hearing aid.

Dr. Gordon D. Hoople, Syracuse, N. Y.: I speak from a neutral standpoint. I am not an expert fitter of hearing aids and I have not done the fenestration operation.

To make a just comparison with Dr. Campbell's series, the result of the use of some badly fitted and outmoded hearing aids should have been included. Dr. Shambaugh says that all but 2 of his patients who received permanent improvement of hearing now claim that they hear better without their hearing aids than they did with them. In reporting results with modern, well fitted hearing aids, a proper comparison should be made. Favorable results other than the increase of hearing should be considered, such as the loss of tinnitus and the intelligibility of conversation in a group.

I believe the fenestration operation should be in the hands of a few specially trained and remarkably skilful men. The training should include not only technic but the proper selection of cases. We all know of patients for whom the operation has accomplished nothing. I have knowledge of doctors who are doing this operation and are not qualified and others who are contemplating including it in their surgical practice. I believe the end results will not be known for another five or ten years. In the meantime those who are especially skilful should continue to operate in properly selected cases.

Dr. Gordon Berry, Worcester, Mass.: I should like to have Mr. Watson of the Maico Company, clarify the point made by Dr. Bunch as to how these audiograms were made.

Mr. Watson: These tests were made by using a loud speaker in free air in a soundproof room, measuring two factors—the gain in amplification and the sensitivity. (The technical details may be read in the original discussion.)

Dr. Edmund P. Fowler, New York: The people who want to use a hearing aid do not want the operation and will not have it. The people who want the operation will not wear a hearing aid. Many people want the operation because they will not wear a hearing aid. That is nothing against the operation. It is worth while and will be eventually worked out, but there has been at times a skimming around the facts on both sides.

Patients have told me that salesmen for manufacturers of hearing aids told them that they were going to lose their hearing if they did not wear a hearing aid—these salesmen working on commission. On the other hand, patients have been told that the operation would save their hearing, which would otherwise get worse. All otologists know that otosclerosis frequently comes to rest with or without operation. The effect of the operation on the increase of otosclerosis is most important and if more data can be obtained on this, it will be a most valuable outcome.

Dr. Gordon Berry, Worcester, Mass.: Dr. Bunch brought out an important point. It is the final hearing which is important and not the amount of gain. I wish to clarify one point in Dr. Hoople's suggestion. I have carefully compared Dr. Lempert's selected cases with the selected cases in which hearing aids were fitted by Mr. Watson. That is, I took selected cases in both groups. I think a comparison could possibly be made with the Council of Physical Therapy's hearing aids which give a decibel gain.

I am grateful to Mr. Watson for explaining how the charts were made and to Dr. Fowler for bringing out the important point as to whether the operation influences the progress of the disease.

Book Reviews

Ozena (Rinite atrópica fétida). Ernesto Moreira. Privately printed. São Paulo, Brazil, 1942.

In a detailed monograph, Moreira surveys the problem of ozena in Brazil.. Over a period of twenty years the incidence was 3.8 per cent of all patients treated for rhinologic conditions. The disease was encountered more frequently in females than in males, and its occurrence favored the second decade of life.

Heredity acted as a factor merely in transmitting certain predispositions to ozena, but not in transmitting the disease itself. Climatic influences seemed to play a role in its incidence, the disease being encountered more frequently in the mountainous regions, and rarely in the maritime areas. It occurred in all social classes, and contagion was not a factor.

The author elaborates on the various etiologic factors, such as thyroid, ovarian and orchitic dysfunctions. Toxic neuritis and metabolic disturbances are also discussed.

The symptomatology and diagnosis—on the basis of clinical, roentgenologic and laboratory data—are described, and, lastly, therapeusis, both medical and surgical, is considered. Moreira employs a modification of the Lautenschläger procedure, which he discusses in detail. The essential point to be stressed in this modification is a special form of suture for the fixation of the mobilized lateral nasal walls. The author reports 100 cases in which this technic was employed; the results were excellent in 48 per cent, good in 29 per cent and fair in 21 per cent, and there was no improvement in 2 per cent.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in New York, at the Waldorf-Astoria Hotel and the New York Eye and Ear Infirmatory, on June 3, 4 and 5, 1943. Candidates should apply at once to Dr. Dean M. Lierle, secretary of the board, University Hospital, Iowa City.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James G. Dwyer, 375 Park Ave., New York.
President-Elect: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Edward J. Whalen, 750 Main St., Hartford, Conn.
Southern.—Chairman: Dr. Francis B. Blackmar, Swift Kyle Bldg., Columbus, Ga.
Middle.—Chairman: Dr. Voss Harrell, Hofman Bldg., Detroit.
Western.—Chairman: Dr. Irving M. Lupton, Medical Arts Bldg., Portland, Ore.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.